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Health and Environmental Effects Profile for Pyrene

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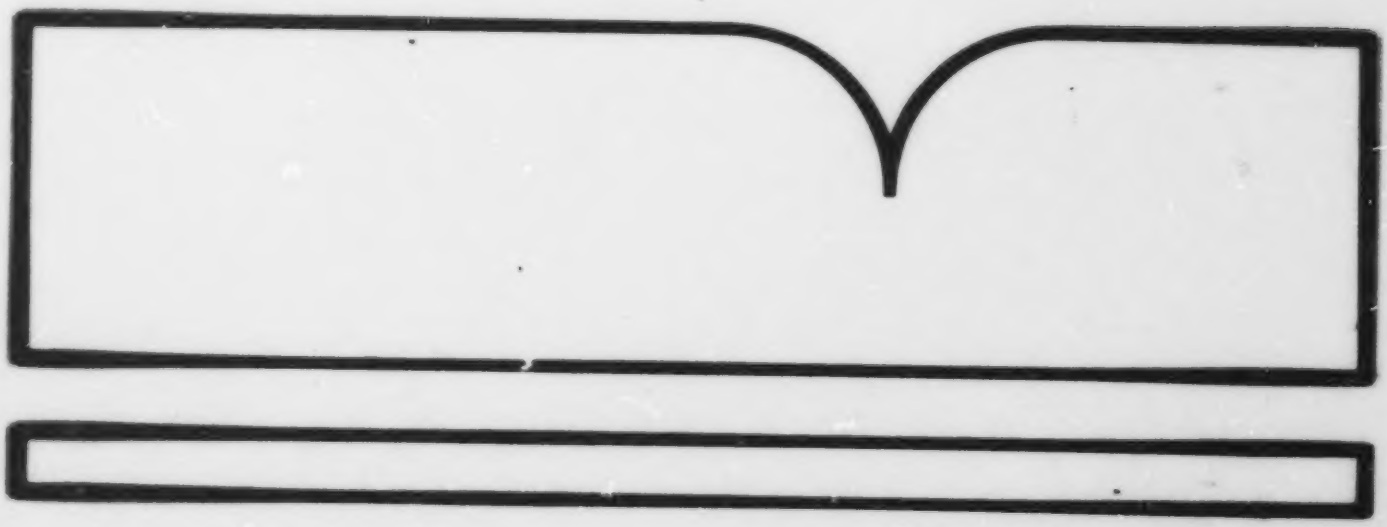

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16. ABSTRACT

The Health and Environmental Effects Profile for Pyrene was prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA) and to provide health-related limits for emergency actions under Section 101 of the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA). Both published literature and information obtained from Agency program office files were evaluated as they pertained to potential human health, aquatic life and environmental effects of hazardous waste constituents.

Quantitative estimates have been presented provided sufficient data are available. Existing data are insufficient to determine a Reference Dose (RfD) or a carcinogenic potency factor for pyrene.

The Reportable Quantity (RQ) value of 1, 10, 100, 1000 or 5000 pounds is used to determine the quantity of a hazardous substance for which notification is required in the event of a release as specified by CERCLA based on chronic toxicity. Existing data are insufficient to determine an RQ value.

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HEALTH AND ENVIRONMENTAL EFFECTS PROFILE FOR PYRENE

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PREFACE

Health and Environmental Effects Profiles (HEEPs) are prepared for the Office of Solid Waste and Emergency Response by the Office of Health and Environmental Assessment. The HEEPs are intended to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA), as well as to provide health-related limits for emergency actions under Section 101 of the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA). Both published literature and information obtained from Agency program office files are evaluated as they pertain to potential human health, aquatic life and environmental effects of hazardous waste constituents. The literature searched and the dates of the searches are included in the section titled "Appendix: Literature Searched." The literature search material is current through November, 1985.

Quantitative estimates are presented provided sufficient data are available. For systemic toxicants, these include Reference doses (RfDs) for chronic exposures. An RfD is defined as the amount of a chemical to which humans can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect. In the case of suspected carcinogens, RfDs are not estimated in this document series. Instead, a carcinogenic potency factor of q_1^* is provided. These potency estimates are derived for both oral and inhalation exposures where possible. In addition, unit risk estimates for air and drinking water are presented based on inhalation and oral data, respectively.

Reportable quantities (RQs) based on both chronic toxicity and carcinogenicity are derived. The RQ is used to determine the quantity of a hazardous substance for which notification is required in the event of a release as specified under CERCLA. These two RQs (chronic toxicity and carcinogenicity) represent two of six scores developed (the remaining four reflect ignitability, reactivity, aquatic toxicity and acute mammalian toxicity).

The first draft of this document was prepared by Syracuse Research Corporation under EPA Contract No. 68-03-3228. The document was subsequently revised after reviews by staff within the Office of Health and Environmental Assessment: Carcinogen Assessment Group, Reproductive Effects Assessment Group, Exposure Assessment Group, and the Environmental Criteria and Assessment Office in Cincinnati.

The HEEPs will become part of the EPA RCRA and CERCLA dockets.

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EXECUTIVE SUMMARY

Pure pyrene is a colorless solid at ambient temperatures. The presence of tetracene, a common impurity, gives yellow color to pyrene. It is fairly soluble in organic solvents including ethanol, ethyl ether and benzene (Windholz, 1983; Weast, 1980), but is almost insoluble in water (Pearlman et al., 1984). Chemically, pyrene is susceptible to oxidation by ozone, peroxides and other oxidants (NAS, 1972). Currently, pyrene is neither commercially produced nor imported into the United States (SRI, 1986). It can be isolated from coal tar or from the products of destructive hydrogenation of coal (Windholz, 1983). There is no known commercial use of pyrene (IARC, 1983).

If released to the aquatic environment, adsorption to particulate organic matter and sediments in water will be an important environmental fate process based on measured sediment K_{oc} values and widespread monitoring of pyrene in ambient sediments. In the dissolved state in the water column, direct photolysis may be significant, as the average photolytic half-life of pyrene near the surface at 40°N latitude has been estimated to be ~1.1 hours in June and 3.1 hours in January (Zepp and Schlotzhauer, 1983). In deep, turbid water, photolysis may not be an important process. Microbial degradation data (Walker and Colwell, 1975) suggest that pyrene may be susceptible to biodegradation in natural water. Microbial oxidation of PAH requires oxygen and will not proceed in anoxic sediments or water (U.S. EPA/NIH, 1986). In clear and shallow water, biodegradation will not be important. It will, however, be an important process in slow moving, deep and turbid waters. Volatilization may have some significance in shallow, rapidly flowing rivers. Bioaccumulation potential appears to be

dependent on the organism being considered. Hydrolysis is not expected to be important. The detection of a nearly constant concentration of pyrene in sediment cores of a remote forest lake (Tan and Heit, 1981) indicate that pyrene is very persistent under anaerobic and dark conditions. If released to the atmosphere, pyrene will exist in both vapor and particulate-adsorbed phases; monitoring data (Ligocki et al., 1985a,b; Yamasaki et al., 1982) suggest that the vapor-phase may dominate. Vapor-phase pyrene appears to be susceptible to relatively rapid decomposition by direct photolysis or reaction with photochemically produced HO radical. Adsorption of pyrene to particulates containing a high carbon content significantly attenuates photolysis and other chemical oxidation reactions (Behymer and Hites, 1985; Valerio and Lazzarotto, 1984; Yokley et al., 1986; Santodonato et al., 1981), and may therefore permit the long-range atmospheric transport (Lunde and Bjoerseth, 1977). Pyrene may be physically removed from the atmosphere by wet and dry deposition (Ligocki et al., 1985a,b; Tan and Heit, 1981). If released to soil, pyrene may be susceptible to biodegradation under aerobic conditions. Under most conditions, it is not expected to leach or volatilize significantly and may persist in soils.

Human exposure to pyrene occurs primarily through the inhalation of tobacco smoke and polluted air and by the ingestion of contaminated food and water (IARC, 1983). Pyrene occurs ubiquitously as a product of incomplete combustion and occurs naturally in fossil fuels (IARC, 1983). It has been widely detected in drinking water, surface water, groundwater, rainwater and aquatic sediments (see Tables 3-1 and 3-2), in many foods (Santodonato et al., 1981; Dennis et al., 1983), and in the general ambient atmosphere (see Table 3-3). The average dietary intake of pyrene in England is ~1.09 µg/day (Dennis et al., 1983); the average intake from drinking water in

the United States is ~2.0 ng/day, while the inhalation intake in the United States is ~10-400 ng/day. The presence of pyrene in food is a result of contamination from a polluted environment and formation during cooking by pyrolysis of organic matter (Santodonato et al., 1981; Fazio and Howard, 1983). Emission to the atmosphere results from the combustion of fossil fuels (oil, coal), wood burning for residential heating, gasoline and diesel engine exhaust, and open burning (agricultural burning, forest fires, structure burning, refuse burning) (NRC, 1983).

Of six species tested, three arthropods were more sensitive to pyrene than fish or amphibians. LC_{50} values of 4, 8 and 20 µg/ℓ were reported for Daphnia magna, Artemia salina and Aedes aegypti, respectively. Like some other PAH, the toxicity of pyrene is greatly enhanced by UV light or sunlight (Kagan et al., 1985). Two algae species were less sensitive to pyrene than the three arthropods, with EC_{50} values of 202 and 332 µg/ℓ for inhibition of photosynthesis (Hutchinson et al., 1980). Reported BCF values ranged from 72 for rainbow trout (Gerhart and Carlson, 1978) to 36,300 for the alga, Selenastrum capricornutum (Casserly et al., 1983).

Limited gastrointestinal absorption of pyrene was measured 24 hours after gavage administration of 50 µg pyrene to male rats (Mitchell and Tu, 1977, 1979). Pyrene was detected fluorometrically in the gastrointestinal tract at ~50% of the administered dose; no pyrene was detected in the liver, kidneys, lungs or trachea. An interpretation of poor gastrointestinal absorption of pyrene from these data is supported by detection of pyrene in the liver and kidneys 24 hours after inhalation exposure, but complicated by possible biliary elimination of metabolites since the analytical technique was not specific for pyrene. Furthermore, the method of administration was not environmentally relevant. Rapid absorption of inhaled pyrene by rats is

indicated by fluorometric detection of pyrene in respiratory tissues, liver and kidneys 30 minutes after 1-hour exposure to 500 µg/ℓ pyrene aerosol (Mitchell and Tu, 1979). Distribution to tissues other than the liver and kidney was not assessed in the inhalation study.

In vivo and in vitro studies indicate that metabolism of pyrene proceeds by oxidation at the 1-2 and 4-5 bonds, with 1-hydroxypyrene as the principal metabolite (Boyland and Sims, 1964; Sims, 1970; Jacob et al., 1982; Keimig et al., 1983). The metabolites have been detected as sulfuric acid and glucuronic acid conjugates in the urine of rats, rabbits and pigs. Urinary excretion and fecal elimination of pyrene has not been quantitated, but in limited oral and inhalation studies with rats, fecal elimination appears to reflect unabsorbed pyrene, pyrene resulting from mucociliary clearance or biliary elimination (Mitchell and Tu, 1979).

Intratracheal instillation of 3 mg pyrene suspended in saline at weekly intervals for 30 weeks did not produce tumors in the respiratory system (histologic examination) or other tissues (gross examination) in hamsters observed for life (Sellakumar and Shubik, 1974). Skin tumors were not induced in mice by twice or three-times weekly dermal application of pyrene for 1-2 years (Barry et al., 1935; Badger et al., 1940; Wynder and Hoffmann, 1959; Roe and Grant, 1964; Horton and Christian, 1974; Van Duuren and Goldschmidt, 1976). Mouse skin initiation-promotion studies involving initiation with benzo[a]pyrene (Roe and Grant, 1964), promotion with croton oil (Salaman and Roe, 1956) or promotion with TPA (Scribner, 1973; Wood et al., 1980) were negative or inconclusive (Scribner, 1973). Enhancement of the dermal carcinogenicity of benzo[a]pyrene in mice by simultaneous application of pyrene has, however, been reported (Goldschmidt et al., 1973; Van Duuren and Goldschmidt, 1976), indicating possible cocarcinogenicity of

pyrene. Two subcutaneous injections of 10 mg pyrene crystals 4 months apart did not produce local tumors in mice after 12-18 months (Shear and Leiter, 1941).

Pyrene has been tested extensively in mutagenicity and other short-term assays. Pyrene was mutagenic in some tests with S. typhimurium in the presence of metabolic activation preparation (Kaden et al., 1979; Bridges et al., 1981; Matijasevic and Zeiger, 1985; Sakai et al., 1985) and produced unscheduled DNA synthesis in cultured primary hamster hepatocytes (McQueen et al., 1983) and human fibroblasts (Robinson and Mitchell, 1981), mutation in mouse lymphoma L5178Y cells in vitro (Jotz and Mitchell, 1981), sister chromatid exchanges in CHO (Evans and Mitchell, 1981) and V79 (Popescu et al., 1977) cells in vitro and chromosomal aberrations in Chinese hamster V79 cells in vitro (Popescu et al., 1977). Genotoxicity of pyrene was not demonstrated in other assays using the same or different indicator organisms or endpoints, including morphological transformation in a variety of rat, mouse, hamster and guinea pig systems.

Pertinent data regarding teratogenicity or other reproductive effects of pyrene could not be located in the available literature as cited in the Appendix.

Inhibition of growth and enlarged, fatty livers were reported as effects in a group of six male rats maintained on diets that contained 2000 ppm pyrene for as long as 100 days (White and White, 1939). The significance of these findings is uncertain because incidences were not reported, a small number of animals was tested, the treatment durations were variable and unspecified, and control data were not reported. Pertinent data regarding toxic effects of chronic oral or inhalation exposure to pyrene could not be located in the available literature as cited in the Appendix.

A single intraperitoneal injection of 150 mg/kg pyrene produced minimal gross swelling and congestion of the liver and increased serum AST and bilirubin in rats (Yoshikawa et al., 1985).

Data were insufficient to derive an RfD, q_1^*, RQ or F factor. Pyrene is a U.S. EPA Group D chemical; that is, it cannot be classified as to its human carcinogenic potential.

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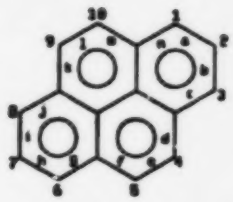
LIST OF ABBREVIATIONS

ADI	Acceptable daily intake
ALT	Alanine aminotransferase
AST	Aspartate aminotransferase
BUN	Blood urea nitrogen
CAS	Chemical Abstract Service
CHO	Chinese hamster ovary
DMSO	Dimethyl sulfoxide
DNA	Deoxyribonucleic acid
EC_{50}	Median effective concentration
GGPT	Gamma-glutamyl transpeptidase
K_{oc}	Soil sorption coefficient
K_{ow}	Octanol water partition coefficient
K_{sed}	Sorption coefficient between sediment and water
LC_{50}	Median lethal concentration
LD_{50}	Median lethal dose
LDH	Lactate dehydrogenase
MED	Minimum effective dose
PAH	Polycyclic aromatic hydrocarbons
ppb	Parts per billion
ppm	Parts per million
ppt	Parts per trillion
RQ	Reportable quantity
TLC	Thin layer chromatography
TLV	Threshold limit value
TPA	Terephthalic acid
TWA	Time-weighted average
UV	Ultraviolet

1. INTRODUCTION

1.1. STRUCTURE AND CAS NUMBER

The chemical commonly called pyrene is also known by the synonym benzo-(d,e,f)phenanthrene. The structure, empirical formula, molecular weight and CAS Registry number for this chemical are as follows:



Empirical formula: $C_{16}H_{10}$

Molecular weight: 202.24

CAS Registry number: 129-00-0

Both the numerical and alphabetical numbering system for the substituents in the ring are indicated in the structure above (Santodonato et al., 1981).

1.2. PHYSICAL AND CHEMICAL PROPERTIES

Pure pyrene is a colorless crystalline solid at ambient temperatures. The presence of tetracene, a common contaminant, gives yellow color to pyrene. It is fairly soluble in organic solvents including ethanol, ethyl ether and benzene (Windholz, 1983; Weast, 1980), but is almost insoluble in water. A few relevant physical properties of pyrene are listed below:

Melting point:	156°C	Windholz, 1983
Boiling point:	404°C	Windholz, 1983
Density at 23°C:	1.271 g/cm³	Windholz, 1983
Solubility in distilled water at 25°C:	0.129-0.156 mg/ℓ 0.138 mg/ℓ (average)	Pearlman et al., 1984
Solubility in Pacific seawater at 20°C:	0.065 mg/ℓ	Hashimoto et al., 1984

Log K_{ow}:	4.88-5.22	Ruepert et al., 1985; Ogata et al., 1984; Yalkowsky and Valvani, 1979; Karickhoff, 1981; Miller et al., 1985
Vapor pressure at 25°C:	6.85×10^{-7} mm Hg	Santodonato et al., 1981
Henry's Law constant: at 25°C	1.1×10^{-5} atm-m³/mol	Mackay et al., 1982

Chemically, PAH are reasonably reactive. They can undergo substitution and addition reactions. In addition, these compounds are susceptible to oxidation by ozone, peroxides and other oxidants (NAS, 1972).

1.3. PRODUCTION DATA

Currently, pyrene is neither commercially produced nor imported into the United States (SRI, 1986). According to TSCA Inventory of chemical producers (U.S. EPA, 1977), two companies imported pyrene into the United States in 1977, but their individual or total import volume was not reported. Pyrene occurs in coal tar. It is also obtained by the destructive hydrogenation of coal (Windholz, 1983).

1.4. USE DATA

There is no known commercial use of pyrene (IARC, 1983). Small amounts of pyrene are used for scientific research (Hawley, 1981).

1.5. SUMMARY

Pure pyrene is a colorless solid at ambient temperatures. The presence of tetracene, a common impurity, gives yellow color to pyrene. It is fairly soluble in organic solvents including ethanol, ethyl ether and benzene (Windholz, 1983; Weast, 1980), but is almost insoluble in water (Pearlman et al., 1984). Chemically, pyrene is susceptible to oxidation by ozone, peroxides and other oxidants (NAS, 1972). Currently, pyrene is neither commercially produced nor imported into the United States (SRI, 1986; USITC,

1984). It can be isolated from coal tar or from the products of destructive hydrogenation of coal (Windholz, 1983). There is no known commercial use of pyrene (IARC, 1983).

2. ENVIRONMENTAL FATE AND TRANSPORT PROCESSES

2.1. WATER

2.1.1. Hydrolysis. Pyrene contains no hydrolyzable functional groups; therefore, aquatic hydrolysis is not expected to be significant (Mabey et al., 1981).

2.1.2. Oxidation. The rate constants for the oxidation of pyrene with photochemically produced RO_2 radical and $^1O_2$ at 25°C have been estimated to be ~2.2×10^4 and 5×10^9 M^{-1} $hour^{-1}$, respectively (Mabey et al., 1981). Assuming the RO_2 radical and $^1O_2$ concentrations of natural water are 10^{-9} and 10^{-12} M (Mabey et al., 1981), respectively, the respective half-lives are ~3.6 years and 58 days. These half-lives apply to the dissolved fraction of pyrene in the water column, and not to the pyrene that is adsorbed to suspended particulates and sediment.

Pyrene reacts readily in aqueous phase with ozone (Butkovic et al., 1983), which has importance in water purification plants that use ozone for disinfection; however, the ozone reaction is not likely to be important in natural waters.

2.1.3. Photolysis. In a methanol/ethanol solvent, pyrene exhibits UV absorption maxima at 305, 318, 333.5, 351.5, 356, 362 and 371.5 nm (IARC, 1983); similar absorption maxima occur in aqueous solution (Schwarz and Wasik, 1976). Therefore, direct photolysis in sunlight is a definite possibility. Zepp et al. (1984) reported the rate constant for the direct photolysis of pyrene in aqueous solution to be 1.0 $hour^{-1}$ at midday sunlight exposure; this corresponds to a half-life of 41.6 minutes. Based on data reported by Zepp and Schlotzhauer (1983), the average photolytic half-life of pyrene near the surface of a clear water body at 40°N latitude is ~1.1 hours during June and 3.1 hours during January.

The data found in Zepp et al. (1984) suggest that direct photolysis of pyrene in the dissolved state in the water column is a significant transformation process; however, photolysis may be significantly attenuated by pyrene adsorption to suspended particulate matter and sediments.

Thus, the photolytic half-life of pyrene in water will increase as the turbidity and depth of water increase. In deep and turbid waters, photolysis may not be a significant process.

2.1.4. Microbial Degradation. Tabak et al. (1981) examined the biodegradability of pyrene in a static culture flask-screening procedure using settled domestic wastewater as microbial inoculum. At 5 ppm concentration, pyrene was found to be significantly degraded with rapid adaptation, as 71% of the initial pyrene was degraded in 7 days and 100% was degraded in 7 days following the addition of a second acclimatized subculture. At 10 ppm, however, only 11% degraded in 7 days and the degradation rate decreased to 0% in 7 days with 3-week-old subcultures. This result indicated that pyrene at this concentration was toxic to microorganisms.

Bacteria isolated from Colgate Creek sediment (near the Chesapeake Bay) and cultured in either oil-contaminated Colgate Creek water or a relatively oil-free Eastern Bay water for 28 days degraded 19.6-22.4% of added pyrene in a shake-flask procedure at 20°C (Walker and Colwell, 1975); 2.0-8.2% degradation was observed using bacteria from Eastern Bay sediment. These results indicate that biodegradation of pyrene in most natural water will be slow and that the degradation will be faster with acclimatized microorganisms.

PAHs with four or more aromatic rings are degraded slowly by microbes and biodegradation is considered to be the ultimate fate process in water

(U.S. EPA/NIH, 1986). The concentrations of bacteria and fungi capable of oxidizing the hydrocarbons are extremely low, however, in all but heavily polluted fresh and marine waters, and most species cannot use PAH as a sole carbon source. Microbial oxidation of PAH requires oxygen and will not proceed in anoxic sediments or water (U.S. EPA/NIH, 1986).

2.1.5. Volatilization. The Henry's Law constant for pyrene has been experimentally determined to be 1.1×10^{-5} atm-m³/mol at 25°C (Mackay et al., 1982). This value of Henry's Law constant suggests that aquatic volatilization may have some significance, but is not likely to be rapid (Lyman et al., 1982). Based on the method outlined in Lyman et al. (1982), the volatilization half-life from a river 1 m deep, flowing at a speed of 1 m/sec at a wind velocity of 3 m/sec is ~4.8 days; volatilization from static water will be significantly slower. Further, adsorption to sediment is expected to attenuate volatilization. Davis et al. (1983) experimentally determined a pyrene volatilization half-life of 49 days from a laboratory-scale waste stabilization pond study; loss of pyrene from the pond from volatilization accounted for only 0.4% of the total transport and fate.

2.1.6. Adsorption. Karickhoff et al. (1979) experimentally examined the adsorption of pyrene to a variety of river and pond sediments. The following average K_{oc} values, based on the organic carbon content of the sediments, were determined: sandy sediments, 19,000; coarse silt, 93,000; medium silt, 130,000; fine silt, 110,000; and clay, 120,000. These K_{oc} values indicate that pyrene will be strongly adsorbed onto sediments. The K_{sed} values varied from 9.4 to 3800 and were strongly influenced by particle size and organic content. Karickhoff and Morris (1985) found that several weeks may be required for complete equilibration of pyrene between sediments and the water column.

The K_{oc} values listed above and the widespread detection of pyrene in U.S. sediments (Section 3.1.) at concentrations much higher than in water columns indicate that adsorption to particulate organic matter and sediments is an important environmental fate process. Movement by sediments is considered to be an important transport process for PAH (U.S. EPA/NIH, 1986).

2.1.7. **Bioconcentration.** The following experimental BCF values have been reported: 72 (rainbow trout, Salmo gairdneri, 21-day exposure, flowing conditions) (Spehar et al., 1980); 457 (goldfish, Carassius auratus) (Ogata et al., 1984); 600-970 (fathead minnow, Pimephales promelas) (Carlson et al., 1979); 2690 (unspecified species) (Mackay, 1982); 2702 (Daphnia pulex, 24-hour) (Southworth et al., 1978); 13,072 (unspecified algae) (Davis et al., 1983).

BCF values >1000 are considered significant (Kenaga, 1980). PAH may not appreciably bioconcentrate in organisms that have microsomal oxidase, such as fish, as this enzyme metabolizes PAH (Santodonato et al., 1981). Therefore, the bioaccumulation potential may be very dependent on the organism being considered.

2.1.8. **Persistence.** Tan and Heit (1981) monitored sediment cores taken from Woods Lake in the remote Adirondack Forest of upstate New York for various PAH. The following pyrene concentrations (ng/g dry sediment weight) were found at various depths: 930 (0-4 cm), 290 (4-8 cm), 56 (8-11 cm), 67 (12-17 cm), 37 (24-26 cm), 60 (34-38 cm), 21 (42-44 cm), 89 (50-54 cm) and 16 (80-84 cm). The near constancy in the concentration of pyrene in the sediment cores deeper than 8 cm indicates that the compound is very persistent, particularly under anaerobic conditions and in the absence of sunlight prevalent in the deeper cores.

2.2. AIR

Pyrene can exist in the ambient atmosphere in both the particulate-associated form and in the vapor-phase. From their ambient air monitoring data from Portland, OR, Ligocki et al. (1985a,b) reported the mean pyrene concentration in the vapor-phase to be 6.8 ng/m^3, while the mean concentration in the particulate-phase was reported to be 0.53 ng/m^3. Yamasaki et al. (1982) monitored the urban air of a Japanese city for an entire year and found the pyrene concentration in the vapor-phase to be ~1 order of magnitude higher than in the particulate phase. The form in which pyrene exists in the atmosphere has a significant bearing on its environmental fate. Pyrene in the particulate-associated form is expected to be significantly more persistent than vapor-phase pyrene.

2.2.1. Photolysis. Pyrene strongly absorbs environmental UV radiation and photolyzes quite rapidly in water in the dissolved state (see Section 2.1.3.); therefore, direct photolysis may be expected to be a significant removal mechanism for pyrene vapor in the atmosphere.

Several investigators have examined the photodegradation of pyrene adsorbed to particulate materials. Behymer and Hites (1985) examined the photodegradation of pyrene while adsorbed to various substrates (silica gel, alumina, flyash, carbon black) by using simulated atmospheric conditions. The following half-lives were determined: silica gel, 21 hours; alumina, 31 hours; flyash, 46 hours; and carbon black, >1000 hours. Adsorption to carbon black clearly stabilized the phototransformation. Valerio and Lazzarotto (1984) found that pyrene photolyzed significantly faster when adsorbed to inorganic components of air particulates as compared with whole atmospheric particulate matter containing particulate organic fractions as well. Yokley et al. (1986) studied the photochemical decomposition of pyrene adsorbed to various fly ashes, alumina, silica gel and glass. Photo-

transformation proceeded more rapidly with the alumina, silica gel or glass as compared with the fly ashes. The high carbon content of the flyashes was effective in suppressing photolysis.

2.2.2. Reaction with HO Radical. The half-life for the vapor-phase reaction of pyrene with photochemically produced HO radical is ~1.12 days at 25°C, assuming an average atmospheric HO radical concentration of 8×10^5 molecules/cm^3 (U.S. EPA, 1986a).

2.2.3. Physical Removal. Removal of pyrene from the atmosphere may occur by wet and dry deposition. Particulate-associated pyrene has been detected in rainwater (Ligocki et al., 1985b). The presence of pyrene in lake sediments in the Adirondack Forest, NY, has been attributed to physical deposition (Tan and Heit, 1981). Dissolved pyrene has also been detected in rainwater (Ligocki et al., 1985a), suggesting that physical removal of vapor by washout or dissolution into clouds with subsequent rainfall may be possible.

Lunde and Bjoerseth (1977) reported that pyrene has been transported in the atmospheric aerosol from England to Norway, indicating that long-range transport of particulate-phase pyrene occurs. This is consistent with the experimental data indicating that the persistence of pyrene is increased when it is is adsorbed to particulate substrate.

2.3. SOIL

2.3.1. Microbial Degradation. Groenewegen and Stolp (1981) examined the degradation of pyrene in a laboratory soil-water percolation study over a 4-week incubation period. Significantly more degradation occurred in the units that were not sterilized by $HgCl_2$. The authors attributed the decline in pyrene concentration in the nonsterilized units to biological breakdown. The microbial data listed in Section 2.1.4. also suggest that pyrene may be susceptible to biodegradation in soil under aerobic conditions.

2.3.2. **Chemical Degradation.** Pertinent data regarding the chemical degradation of pyrene could not be located in the available literature as cited in the Appendix.

2.3.3. **Adsorption.** Karickhoff et al. (1979) reported experimental K_{oc} values ranging from 19,000-130,000 and K_{sed} values of 9.4-3800 (see Section 2.1.6.) for various pond and river sediments. Kenaga (1980) reported an experimental soil K_{oc} value of 84,000. These K_{oc} values suggest soil immobility (Swann et al., 1983); however, the detection of pyrene in several groundwaters (Section 3.1.) indicates that leaching can occur. This leaching may occur in soils containing low organic matter (e.g., sand) or high porosity. Leaching is also possible from spill or waste disposal sites contaminated with materials containing relatively high concentrations of PAH. In addition, Karickhoff and Morris (1985) noted that equilibration of pyrene between sorbed and water-phase may require several weeks; therefore, some movement may occur before equilibrium is reached. Pyrene is not, however, expected to leach in soil under most conditions.

2.3.4. **Volatilization.** Volatilization of pyrene from soils is not expected to be an important process (Sims and Overcash, 1983).

2.4. SUMMARY

If released to the aquatic environment, adsorption to particulate organic matter and sediments in water will be an important environmental fate process based on measured sediment K_{oc} values and widespread monitoring of pyrene in ambient sediments. In the dissolved state in the water column, direct photolysis may be significant, as the average photolytic half-life of pyrene near the surface at 40°N latitude has been estimated to be ~1.1 hours in June and 3.1 hours in January (Zepp and Schlotzhauer, 1983). In deep, turbid water, photolysis may not be an important process.

Microbial degradation data (Walker and Colwell, 1975) suggest that pyrene may be susceptible to biodegradation in natural water. Microbial oxidation of PAH requires oxygen and will not proceed in anoxic sediments or water (U.S. EPA/NIH, 1986). In clear and shallow water, biodegradation will not be important. It will, however, be an important process in slow moving, deep and turbid waters. Volatilization may have some significance in shallow, rapidly flowing rivers. Bioaccumulation potential appears to be dependent on the organism being considered. Hydrolysis is not expected to be important. The detection of a nearly constant concentration of pyrene in sediment cores of a remote forest lake (Tan and Heit, 1981) indicates that pyrene is very persistent under anaerobic and dark conditions. If released to the atmosphere, pyrene will exist in both vapor and particulate-adsorbed phases; monitoring data (Ligocki et al., 1985a,b; Yamasaki et al., 1982) suggest that the vapor-phase may dominate. Vapor-phase pyrene appears to be susceptible to relatively rapid decomposition by direct photolysis or reaction with photochemically produced HO radical. Adsorption of pyrene to particulates with a high carbon content significantly attenuates photolysis and other chemical oxidation reactions (Behymer and Hites, 1985; Valerio and Lazzarotto, 1984; Yokley et al., 1986; Santodonato et al., 1981), and may therefore permit the long-range atmospheric transport (Lunde and Bjoerseth, 1977). Pyrene may be physically removed from the atmosphere by wet and dry deposition (Ligocki et al., 1985a,b; Tan and Heit, 1981). If released to soil, pyrene may be susceptible to biodegradation under aerobic conditions. Under most conditions, it is not expected to leach or volatilize significantly and may persist in soils.

3. EXPOSURE

Human exposure to pyrene occurs primarily through the inhalation of tobacco smoke and polluted air and by the ingestion of contaminated food and water (IARC, 1983) and smoked fish and meats. Pyrene occurs ubiquitously as a product of incomplete combustion and occurs naturally in fossil fuels (IARC, 1983).

3.1. WATER

Table 3-1 lists various pyrene monitoring data for drinking water, groundwater, surface water and rainwater; Table 3-2 lists sediment monitoring data from various U.S. locations. In an analysis of the U.S. EPA STORET data base, pyrene was detected in 5.2% of 1271 effluents and 4.0% of 904 surface waters (Staples et al., 1985). Griest (1980) detected pyrene at concentrations of 23.0 µg/g in the sediment and 4.0 µg/ℓ in the water of an effluent channel from a coking plant. A pyrene concentration of 10 µg/ℓ was detected in the wastewater effluent from a tire manufacturing plant (Jungclaus et al., 1976). Wastewater effluents from two coal coking plants were found to contain pyrene at levels as high as 480 µg/ℓ (Walters and Luthy, 1984). In the preliminary findings of the U.S. EPA Nationwide Urban Runoff Program, pyrene was detected in the stormwater runoff from six U.S. cities at levels ranging from 0.3-10 µg/ℓ (Cole et al., 1984). Hoffman et al. (1984) detected pyrene in both the particulate and soluble phases in urban stormwater runoff feeding the Narragansett Bay watershed.

In general, PAH can be released to water in industrial and municipal effluents, atmospheric fallout and precipitation, road run-off (tire wear, bitumen and asphalt surfaces, cracked lubricating oils) and marine shipping and harbor oil (Santodonato et al., 1981). Sorrell et al. (1980) suggested

TABLE 3-1

Pyrene Monitoring Data for Various Types of Water

Type of Water	Concentration	Location	Sampling Date	Reference
Drinking (finished)	<4.0 ppt	Cincinnati, OH	NR	Sorrell et al., 1980
	<6.0 ppt	Miami, FL		
	<6.0 ppt	Seattle, WA		
	<1.0 ppt	Columbus, OH		
	<1.0 ppt	Cape Girardeau, MO		
	2.0 ppt	Wheeling, WV		
	3.0 ppt	Jefferson Parrish, LA		
	1.0 ppt	Standish, ME		
	1.0 ppt	Ludlaw, MA		
	2.0 ppt	Seattle, WA		
	<1.0 ppt	Colorado Springs, CO		
Drinking	1.4-72.0 ppt	12 Canadian Great Lakes cities	January, 1980	Williams et al., 1982
	ND-31.0 ppt	12 Canadian Great Lakes cities	July-August, 1980	Williams et al., 1982
Drinking	0.53-1.7 ppt	Ottawa, Canada	January-February, 1978	Benoit et al., 1979
Drinking (tap)	<0.30-12 ppt	Norway, Finland, Sweden	NR	Kveseth et al., 1982
Drinking (tap)	1.1 ppt	Norway	NR	IARC, 1983
Groundwater	104.0 ppt	Germany	NR	Sorrell et al., 1980
Groundwater	NR	St. Louis Park, MN	post-1982	Rostad et al., 1985
Groundwater	1.6-2.5 ppt	Germany	NR	IARC, 1983
Surface (river)	NR	Mississippi River (New Orleans, LA)	February, 1979	Overton et al., 1980
	<1.0 ppb	NR, U.S.	1975-1976	Jungclaus et al., 1978
	trace	Delaware River	August, 1977	Hites, 1979
	NR	Niagara River (Niagara Falls)	1979	Elder et al., 1981
Surface (lake)	NR	Lakes Ontario, Erie, Michigan, and Superior	NR	Great Lakes Water Quality Board, 1983
	NR	Lake Pontchartrain, LA	NR	McFall et al., 1979
Surface (river)	5.4 ppb (water and sediment)	Lower Tennessee River	NR	Goodley and Gordon, 1976
	NR	Ohio River System	1978	Ohio River Valley Sanit. Comm., 1979
	18.0 ppt	Tamar Estuary (England)	1980	Readman et al., 1982
	260 ppt	Thames River (England)	NR	Sorrell et al., 1980
	0.3-109.8 ppt	Germany	NR	Sorrell et al., 1980
Rainwater	39 ppt (mean) (dissolved)	Portland, OR	1984	Ligocki et al., 1985a
Rainwater	4.1 ppt (average) (particulate)	Portland, OR	1984	Ligocki et al., 1985b
Rainwater	ND-37 ppt	Rotterdam, Netherlands	September, 1983	Van Noort and Wondergem, 1985

NR = Not reported; ND = not detected

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TABLE 3-2

U.S. Sediment Monitoring Data for Pyrene

Concentration	Location	Sampling Date	Reference
30-31 ng/g (ppt)	Washington State (river bottom sediment)	1979-1980	Prahl et al., 1984
26-40 ng/g (ppt)	Columbia River (suspended sediment)	1979-1980	Prahl et al., 1984
140-280 ng/g (ppt)	Cayuga Lake, NY (deepwater sediment)	1978	Helt, 1985
110-660 ng/g (ppt)	Cayuga Lake, NY (littoral sediment)	1978	Helt, 1985
50-600 ppb	Lake Erie (superficial sediments up to 10 km off coast)	NR	Eadie et al., 1982
930 ng/g (ppt)(0-4 cm depth)	Woods Lake, NY	NR	Tan and Helt, 1981
290 ng/g (ppt)(4-8 cm depth)	Woods Lake, NY	NR	Tan and Helt, 1981
16 ng/g (ppt)(80-84 cm depth)	Woods Lake, NY	NR	Tan and Helt, 1981
ND-1680 ppb	Casco Bay, ME (30 locations)	April, 1980	Larsen et al., 1983
330-18,000 ppb	Eagle Harbor, WA	December, 1983	Malins et al., 1985a
93 ng/g (ppt)	President Point, WA	November 1983	Malins et al., 1985a
2400 ng/g (ppt)	Duwamish River Delta, WA	NR	Varanasi et al., 1985
NR	Elizabeth River, VA	NR	Merrill and Wade, 1985
<1.0-130 ng/g (ppt) (pyrene and fluoranthene)	Georges Bank, Northeastern U.S. Coast	1977; 1981-1982	Boehm and Farrington, 1984
16-539 ppb	Penobscot Bay, ME (49 stations)	1982	Johnson et al., 1985
tract-4000 ng/g (ppt)	Niagara River (suspended sediment)	NR	Kuntz, 1984

NR = Not reported; ND = not detected

that pyrene and other PAHs may contaminate drinking water supplies as a result of coal-tar or asphaltic materials used to line pipes and storage tanks.

Based on the monitoring data from Table 3-1, the concentration of pyrene in drinking water may be ~1.0 ppt (ng/ℓ). Assuming an average daily water intake of 2.0 ℓ, the intake of pyrene in drinking water is ~2.0 ng/day.

3.2. FOOD

In general, PAH found in food are present as a result of contamination from a polluted environment or are formed during the cooking process (Santodonato et al., 1981; Fazio and Howard, 1983); minute amounts of geochemical or biosynthetic origin may also be present (Fazio and Howard, 1983).

Fazio and Howard (1983) reported the detection of pyrene in the following foods: vegetable oils (0.4-5.8 µg/kg), oysters (0.9-6.5 µg/kg), dark coffee (2-8 µg/kg), horse mackerel (0.2-7.0 µg/kg), charcoal-broiled steaks (0.9 µg/kg) and barbecued ribs (1.4 µg/kg). Lawrence and Weber (1984) identified the following concentrations of pyrene in Canadian foods: breakfast cereals (2.2-21 µg/kg), flour (2.6-5.7 µg/kg), skim milk powder (<0.1-2.1 µg/kg) and cooking oils (<0.1-1.4 µg/kg).

Dennis et al. (1983) examined total-diet samples of food groups in England and found the following mean pyrene concentrations (in µg/kg) in the various food groups: cereals (1.85), meat (0.55), fish (0.79), oils and fats (2.75), fruit and sugar (0.83), vegetables (0.24-1.23), beverages (0.04) and milk (0.04). Dennis et al. (1983) then estimated the total pyrene dietary load to be 1.09 µg/person/day. Based on monitoring of total-diet samples collected in the Netherlands, Vaessen et al. (1984) estimated the median intake of pyrene to be 2.2 µg/person/day.

3.3. INHALATION

Table 3-3 lists recent (1979-1983) U.S. ambient air monitoring data for pyrene. It is possible that the atmospheric load of pyrene has been generally decreasing over the past 40 years (Santodonato et al., 1981). This decrease may be a result of decreases in coal consumption for residential heating and industrial uses, improved disposal methods of solid wastes, restrictions on open burning, and improved efficiencies for stationary incineration and combustion sources with improvements of pollution control. The higher atmospheric levels of pyrene in wintertime versus summertime air in New Jersey (see Table 3-3) may be a reflection of an increased use of fossil fuel combustion for heating purposes.

In general, PAHs are emitted to the atmosphere by the combustion of fossil fuels (oil, coal), in exhaust from gasoline and diesel engines, by open burning (agricultural burning, forest fires, structure fires, refuse burning) and by the burning of wood, especially for residential heating (NRC, 1983).

Pyrene has been identified in mainstream cigarette smoke (0.017-27 µg/cigarette), sidestream cigarette smoke (101.1-390 µg/cigarette), smoke-filled rooms (66 ng/m^3), cigar and pipe smoke, mainstream marijuana smoke (0.066 µg/cigarette), gasoline engine exhaust, exhaust from burnt coal and coal-tar (IARC, 1983). The concentration of pyrene detected in various fly-ash samples ranged from 0.5-120 ng/g (Eiceman et al., 1981).

Assuming that the approximate average ambient air concentration of pyrene (gas-phase and particulates) ranges from 0.5-20 ng/m^3 (see Table 3-3) and assuming a human air intake of 20 m^3/day, the average daily intake for an adult can be ~10-400 ng. Matsumoto and Kashimoto (1985) used Japanese monitoring data to estimate an average daily intake of 17 ng in Japan.

TABLE 3-3

U.S. Air Monitoring Data for Pyrene 1979-1984

Concentration (ng/m³)	Location	Sampling Date	Reference
0.041 (particulate)	Quillayute, WA	1979	Prahl et al., 1984
0.029 (particulate)	Sequim, WA	1979	Prahl et al., 1984
0.38 (particulate)	Seattle, WA	1979	Prahl et al., 1984
6.8 (gas-phase) (mean concentration)	Portland, OR	1984	Ligocki et al., 1985a
0.53 (particulates) (average concentration)	Portland, OR	1984	Ligocki et al., 1985b
0.08-0.35 (particulate) (mean)	4 cities in New Jersey	summer, 1982	Greenberg et al., 1985
0.43-2.77 (particulate) (mean)	4 cities in New Jersey	winter, 1983	Greenberg et al., 1985
8 (gas-phase and particulates)	Jacksonville, FL	NR	Kerkhoff et al., 1985
2.2-23 (gas-phase and particulates)	Columbia, SC	1981-1982	Keller and Bidleman, 1984
0.5-4 (gas-phase and particulates)	rural SC (Savannah River)	1982	Keller and Bidleman, 1984

NR = Not reported

3.4. DERMAL

Pertinent data regarding dermal exposure to pyrene could not be located in the available literature as cited in the Appendix.

3.5. SUMMARY

Human exposure to pyrene occurs primarily through the inhalation of tobacco smoke and polluted air and by the ingestion of contaminated food and water (IARC, 1983). Pyrene occurs ubiquitously as a product of incomplete combustion and occurs naturally in fossil fuels (IARC, 1983). It has been widely detected in drinking water, surface water, groundwater, rainwater and aquatic sediments (see Tables 3-1 and 3-2), in many foods (Santodonato et al., 1981; Dennis et al., 1983), and in the general ambient atmosphere (see Table 3-3). The average dietary intake of pyrene in England is ~1.09 µg/day (Dennis et al., 1983); the average intake in drinking water in the United States is ~2.0 ng/day, while the inhalation intake in the United States is ~10-400 ng/day. The presence of pyrene in food is a result of contamination from a polluted environment and formation during cooking by pyrolysis of organic matter (Santodonato et al., 1981; Fazio and Howard, 1983). Emission to the atmosphere results from the combustion of fossil fuels (oil, coal), wood burning for residential heating, gasoline and diesel engine exhaust, and open burning (agricultural burning, forest fires, structure burning, refuse burning) (NRC, 1983).

4. PHARMACOKINETICS

4.1. ABSORPTION

Gastrointestinal absorption of pyrene was evaluated following gavage administration of 50 µg of pyrene in gelatin-saline solution to two male Fischer 344 rats (Mitchell and Tu, 1977, 1979). The rats were killed 24 hours later and the gastrointestinal tract and other tissues were removed. Spectrometric analysis for pyrene in these tissues showed that the gastrointestinal tract contained approximately half of the material that was administered, but showed no significant increase of pyrene in the lung, kidney, liver or trachea. These data were interpreted as suggesting poor gastrointestinal absorption of pyrene, particularly since pyrene was detected in the same tissues 24 hours following inhalation exposure (Section 4.2.). The fluorescence spectra of the major pyrene metabolites, however, overlap those of pyrene; thus, significant quantities of metabolized pyrene in bile can be excreted into the intestine and may not have been detected. Furthermore administration by gavage of pyrene as a suspension of unknown particle size does not preclude absorption by a more relevant route. Also, PAH in general are highly lipid soluble and are absorbed readily from the gastrointestinal tract and lungs (U.S. EPA, 1980).

Significant concentrations of pryrene were detected by fluorescence in the nasal turbinates, trachea, lungs, kidneys and liver of male Fischer 344 rats 30 minutes after exposure to pyrene aerosol (500 µg/ℓ, 0.3-0.8 µm mass median diameter) for 1 hour (Mitchell and Tu, 1979). Although this indicates rapid pulmonary uptake of pyrene, data indicating the extent of uptake were not reported.

4.2. DISTRIBUTION

Rapid distribution of pyrene in rats following inhalation absorption was indicated in the study described in Section 4.1. (Mitchell and Tu, 1979) by

fluorometric detection of pyrene in the kidney, liver, carcass and muscle 30 minutes after exposure. Analyses conducted 1, 2 and 4 days after exposure showed that pyrene was rapidly cleared from these tissues; levels in the lungs were ~65 and 6% of the initial (30-minute) concentrations after 24 and 48 hours, respectively, and concentrations in the liver and kidney were ~6 and 14% of the initial levels, respectively, after 48 hours. Concentrations in the gastrointestinal tract 24 hours after exposure were ~4 times higher than the initial value, indicating clearance from the respiratory tract by mucociliary action. Biliary contribution to gastrointestinal concentrations was not evaluated, but pyrene was largely cleared from the gastrointestinal tract after 4 days.

4.3. METABOLISM

Metabolism of pyrene appears to proceed by oxidation at the 1-2 and 4-5 bonds with 1-hydroxypyrene as the principal metabolite. The sulfuric acid and glucuronic acid conjugates of 1-hydroxypyrene, 1,6- and 1,8-dihydroxypyrene and trans-4,5-dihydro-4,5-dihydroxypyrene, as well as N-acetyl-S-(4,5-dihydro-4-hydroxy-5-pyrenyl)-L-cysteine, were detected in the urine of rats and rabbits that were treated intraperitoneally with pyrene (Boyland and Sims, 1964). N-acetyl-S-(4,5-dihydro-4-hydroxy-5-pyrenyl) L-cysteine and the corresponding cysteinylglycine and glutathione derivatives, the glucuronic acid conjugates of 1-hydroxypyrene and trans-4,5-dihydro-4,5-dihydroxypyrene were also detected in the bile of intraperitoneally-treated rats (Boyland and Sims, 1964). Metabolites isolated following incubation of pyrene with rat-liver microsomes included two uncharacterized trihydroxy derivatives (Jacob et al., 1982) as well as the 1-hydroxy, 1,6-dihydroxy, 1,8-dihydroxy and/or 4,5-dihydrodiol derivatives (Jacob et al., 1982; Sims, 1970). 1-Hydroxypyrene has also been detected in the urine of pigs (~14 kg) that were given single oral doses (1 mg-1 g) of pyrene (Keimig et al., 1983).

4.4. EXCRETION

The oral and inhalation experiments with rats (see Sections 4.1. and 4.2.) indicate that pyrene is eliminated primarily by the gastrointestinal tract (Mitchell and Tu, 1979). It appears that gastrointestinal absorption of pyrene is relatively poor, that inhaled pyrene is cleared to the gut by mucociliary action and that absorbed pyrene and its metabolites are eliminated in the bile. Elimination/excretion was not quantified, as fecal and urine analyses were not conducted.

4.5. SUMMARY

Poor gastrointestinal absorption of pyrene was suggested from the results of a study in which tissues of two male rats were analyzed 24 hours after gavage administration of 50 µg pyrene (Mitchell and Tu, 1977, 1979). Pyrene was detected fluormetrically in the gastrointestinal tract at ~50% of the administered dose, but not in the liver, kidneys, lungs or trachea. This interpretation of poor gastrointestinal absorption of pyrene from these data is further supported by detection of pyrene in the liver and kidneys 24 hours after inhalation exposure, and complicated by possible biliary elimination of pyrene metabolites into the intestinal tract since the analytical technique was not specific for pyrene. Rapid absorption of inhaled pyrene by rats is indicated by fluorometric detection of pyrene in respiratory tissues, liver and kidneys 30 minutes after 1-hour exposure to 500 µg/ℓ pyrene aerosol (Mitchell and Tu, 1979). Distribution to tissues other than the liver and kidney was not assessed in the inhalation study.

In vivo and in vitro studies indicate that metabolism of pyrene proceeds by oxidation at the 1-2 and 4-5 bonds, with 1-hydroxypyrene as the principal metabolite (Boyland and Sims, 1964; Sims, 1970; Jacob et al., 1982; Keimig et al., 1983). The metabolites have been detected as sulfuric acid and

glucuronic acid in the urine of rats, rabbits and pigs. Urinary excretion and fecal elimination of pyrene has not been quantitated, but in limited oral and inhalation studies with rats, fecal elimination appears to reflect unabsorbed pyrene, pyrene resulting from mucociliary clearance and biliary elimination (Mitchell and Tu, 1979).

5. EFFECTS

5.1. CARCINOGENICITY

Studies evaluating the tumorigenic potential of orally-administered pyrene could not be located in the available literature as cited in the Appendix.

A finely aggregated dust mixture containing 3 mg of TLC-purified (>99%) pyrene and an equal amount of hematite (Fe_2O_3, 94% of particles <1.0 μ) suspended in saline was administered to 48 male Syrian golden hamsters (9-10 weeks old) by intratracheal instillation at weekly intervals for 30 weeks (Sellakumar and Shubik, 1974). Ninety untreated hamsters served as controls. The animals were maintained until natural death or sacrificed when moribund, and tissue from the lungs, larynx, trachea and stem bronchi were examined histologically; other organs were examined if gross pathology was present. Survival was 50, 25 and 0% after 50, 80 and 120 weeks, respectively, which was comparable with that of the control group. Respiratory tumors occurred in 1/48 treated hamsters (unspecified tracheal tumor) and in none of the controls (effective number, 82). Two malignant lymphomas also occurred in the treated hamsters, but those also were not treatment-related. A high incidence (39/44) of respiratory tract (primarily bronchi) carcinomas developed in hamsters that were similarly treated with dibenz[a,i]pyrene.

Carcinogenicity studies involving dermal application and subcutaneous injection of pyrene with mice are summarized in Table 5-1. Skin tumors were not induced by dermal application of pyrene 2 or 3 times/week for 1-2 years (Barry et al., 1935; Badger et al., 1940; Wynder and Hoffmann, 1959; Roe and Grant, 1964; Horton and Christian, 1974; Van Duuren and Goldschmidt, 1976).

TABLE 5-1

Dermal and Injection Carcinogenicity Studies of Pyrene

Route	Species/ Strain	No./Sex*	Purity	Treatment	Duration	Effects/Comments	Reference
Skin	mouse/NR	20/NR	purified crude	0.3 g in benzene, twice weekly for life	600 days	1 papilloma; 8/20 survived 1 year; no control data	Barry et al., 1935
Skin	mouse/NR	20/NR	"pure"	0.3 g in benzene, twice weekly for life	717 days	1 papilloma; 5/20 survived 1 year; no control data	Barry et al., 1935
Skin	mouse/NR	40/NR	synthesis	0.3% in benzene twice/week; volume applied not specified	NR	No skin lesions reported; the longest observation time was 680 days; primary report not available	Badger et al., 1940
Skin	mouse/NR	NR/NR	NR	5% in unspecified solvent 3 times weekly for 1 year; volume applied not specified	NR	No skin tumors; primary report not available	Roe and Grant, 1964
Skin	mouse/Swiss	5/f	NR	dose not specified; 10% solution in acetone 3 times weekly for life	12-19 months	No skin tumors; benzo[a]pyrene induced high incidences of skin papillomas and carcinomas under the same conditions	Wynder and Hoffmann, 1959
Skin	mouse/C3H	20/M	high	50 mg in decalin twice weekly for 82 weeks	82 weeks	Skin papillomas and carcinomas in 3/13 and 0/13, respectively; incidences in survivors at 52 weeks; no vehicle controls	Horton and Christian, 1974
Skin	mouse/C3H	20/M	high	50 mg in 50:50 n-dodecane: decalin twice weekly for 82 weeks	82 weeks	Skin papillomas and carcinomas in 2/13 and 2/13, respectively; papillomas in 2/13 vehicle controls; incidences in survivors at 52 weeks	Horton and Christian, 1974
Skin	mouse/ICR/ Ha Swiss	50/f	recrystal-lized	12 or 40 µg in acetone 3 times weekly for 368 or 440 days, respectively	368 or 440 days	No skin papillomas in treated or acetone control groups	Van Duuren and Goldschmidt, 1976
Skin	mouse/NR	NR/NR	NR	unspecified dose of benzo[a]-pyrene followed by 5% pyrene in unspecified solvent for 1 year; application schedule not specified	1 year	No skin tumors; primary report not available	Roe and Grant, 1964
Skin	mouse/S	20/NR	NR	0.3 ml of 0.3% solution in acetone 3 times weekly for 10 applications; starting 25 days later, 18 weekly applications of croton oil in acetone (0.3 ml) consisting of one of 0.17%, two of 0.085% and 15 of 0.17%	25 weeks	Skin papillomas in 6/20 (9 total tumors) versus 4/19 (4 total tumors) in croton oil controls	Salaman and Roe, 1956

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TABLE 5-1 (cont.)

Route	Species/ Strain	No./Sex*	Purity	Treatment	Duration	Effects/Comments	Reference
Skin	mouse/CD-1	30/F	TLC purified	single application of 10 μmol in acetone followed 1 week later by twice weekly applications of 5 μmol TPA for 34 weeks	35 weeks	Skin papillomas in 5/29 (17%) versus 0/30 in controls treated with TPA alone	Scribner, 1973
Skin	mouse/CD-1	30/F	<98%	single application of 0.1 or 0.4 μmol in acetone followed 1 week later by twice weekly applications of 16 μmol TPA for 26 weeks	27 weeks	Skin tumors in 14% (0.1 μmol) and 10% (0.4 μmol) of mice versus 10% in acetone controls; numbers examined not specifically reported but ≥27	Wood et al., 1980
Skin	mouse/ICR/ Ha	50/F	high	applied simultaneously with 5 μg benzo[a]pyrene in acetone at molar ratios (pyrene:B[a]P) of 1:1 or 3:1, 3 times weekly for 33 weeks	33 weeks	Pyrene enhanced skin tumorigenicity of benzo[a]pyrene at 3:1 molar ratio; 13/50 papillomas and 5/50 carcinomas versus 6/50 papillomas and 0/50 tumors with pyrene alone; reported as an abstract	Goldschmidt et al., 1973
Skin	mouse/ICR/ Ha Swiss	50/F	recrystallized	4, 12 or 40 μg with 5 μg benzo[a]pyrene in acetone 3 times weekly for 368, 368 or 440 days, respectively	368 or 440 days	Pyrene enhanced skin tumorigenicity of benzo[a]pyrene at two higher doses; incidences of papillomas and carcinomas were 12/50 and 16/50, 26/50 and 20/50 and 35/50 and 26/50 in the 4, 12 and 40 μg groups, respectively; incidences in benzo[a]pyrene controls were 14/50 and 10/50 (368 days) and 16/50 and 12/50 (440 days); no skin tumors in 50 solvent or untreated controls	Van Duuren and Goldschmidt, 1976
s.c.	mouse/ Jackson A	10/M, 10/F	NR	10 mg pyrene crystals moistened with glycerol injection and repeated after 4 months	18 months	No subcutaneous tumors; average number of pulmonary tumors was 1.6; survival after 12, 15 and 18 months was 23, 13 and 9, respectively	Shear and Leiter, 1941
	mouse/ Strain A	NR/F	NR	12 mg in sunflower oil to pregnant mice on days 18-19 of gestation	offspring observed for 1 year after weaning	No increased incidence of mammary or lung tumors in pyrene-treated or sunflower oil treated compared with untreated control	Nikonova, 1977

*Numbers in treated and control (if used) group(s) unless specified otherwise

NR = Not reported; s.c. = subcutaneous

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Mouse-skin initiation-promotion assays involving initiation with benzo[a]-pyrene (Roe and Grant, 1964), promotion with croton oil (Salaman and Roe, 1956) or promotion with TPA (Scribner, 1973; Wood et al., 1980) were negative or inconclusive (Scribner, 1973). Although pyrene does not appear to be active as a complete carcinogen or initiator or promoter in mouse skin application studies, enhancement of the dermal carcinogenicity of benzo[a]-pyrene in mice by simultaneous application of pyrene has been reported (Goldschmidt et al., 1973; Van Duuren and Goldschmidt, 1976), indicating possible cocarcinogenicity of pyrene. Two subcutaneous injections of pyrene crystals (10 mg) moistened with glycerol, 4 months apart, did not produce local tumors in mice after 12-18 months (Shear and Leiter, 1941), but this study should be regarded as inconclusive because of an inadequate dosing schedule. Subcutaneous administration of 12 mg pyrene to Strain A mice on days 18 and 19 of pregnancy did not increase lung or mammary gland tumor incidences in the progeny 1 year after weaning (Nikonova, 1977).

5.2. MUTAGENICITY

Pyrene has been tested in numerous mutagenicity and other short-term assays with equivocal results. The results of assays conducted before 1983 were summarized by IARC (1983) and were not reviewed independently for this report. Pyrene was reported to be mutagenic in Salmonella typhimurium strains TA97, TA98, TA100, TA1537 and TM677 in the presence of an exogenous metabolic activating system (Kaden et al., 1979; Bridges et al., 1981; Matljasevic and Zeiger, 1985; Sakai et al., 1985), but other reports indicate nonmutagenicity in strains TA1535, TA1537, TA98 and TA100 (McCann et al., 1975; LaVoie et al., 1979; Ho et al., 1981). Pyrene produced unscheduled DNA synthesis in cultured primary hamster hepatocytes (McQueen et al., 1983) and cultured human fibroblast WI38 cells (Robinson and

Mitchell, 1981), but not in cultured primary mouse hepatocytes (McQueen et al., 1983), primary rat hepatocytes, human foreskin epithelial cells or HeLa cells (IARC, 1983). DNA damage, as indicated by SfiA gene expression, was induced in Escherichia coli (Quillardet et al., 1985). Mutation to trifluorothymidine resistance was induced in mouse lymphoma L5178Y cells in vitro (Jotz and Mitchell, 1981). Sister chromatid exchanges were produced in CHO cells (Evans and Mitchell, 1981) and Chinese hamster V79 cells (Popescu et al., 1977) by pyrene in vitro, but this effect was not observed in other studies with CHO cells in vitro (IARC, 1983; Darroudi and Natarajan, 1985), rat liver epithelial ARL 18 cells in vitro (Ved Brat et al., 1983; IARC, 1983) or mouse bone-marrow cells in vivo (IARC, 1983). In vivo treatment with pyrene did not produce sperm head abnormalities in mice (Topham, 1980). Pyrene reportedly produced chromosomal aberrations in Chinese hamster V79 cells in vitro in the presence of a feeder layer (Popescu et al., 1977), but not in rat liver RL cells in vitro in the absence of exogenous metabolic activation (Dean, 1981).

Pyrene was not differentially toxic to DNA-repair-proficient-deficient strains of bacteria (Mamber et al., 1983; IARC, 1983) or CHO cells (Hoy et al., 1984), did not induce prophage in E. coli K12 (Mamber et al., 1984), did not produce DNA single-strand breaks in rat hepatocytes (Sina et al., 1983), was not mutagenic to Saccharomyces cerevisiae or Schizosaccharomyces pombe yeasts (various endpoints examined) (IARC, 1983), was not mutagenic at the HGPRT locus in rat liver ARL18 epithelial cells (Ved Brat et al., 1983), and did not induce sex-linked recessive lethal mutations in Drosophila melanogaster (Valencia and Houtchens, 1981). Pyrene did not induce growth in soft agar of rat ARL liver epithelial cells (Shimada et al., 1983), in pulmonary or peritoneal macrophages from rats treated in vivo (Nielsen and

Anderson, 1986), or in Syrian hamster embryo cells, mouse prostate C3H cells, mouse BALB/C-3T3 cells or guinea pig fetal cells following *in vivo* treatment (IARC, 1983).

5.3. TERATOGENICITY

Pertinent data regarding the teratogenicity of pyrene could not be located in the available literature as cited in the Appendix.

Weaver and Gibson (1979) exposed pregnant rats to graded airborne concentrations of uncharacterized oil shale containing PAH, including pyrene, on days 6-15 of gestation. No treatment-related teratogenic effects were observed during examination of fetuses obtained by Caesarean section on day 20 of gestation. Because of the uncharacterized nature of the test material, it is not possible to quantify the data from this study for use in risk assessment.

5.4. OTHER REPRODUCTIVE EFFECTS

Pertinent data regarding other reproductive effects of pyrene could not be located in the available literature as cited in the Appendix.

5.5. CHRONIC AND SUBCHRONIC TOXICITY

Six male rats of unspecified strain that weighed 75-85 g were maintained on diets that contained 2000 ppm pyrene for periods as long as 100 days (White and White, 1939). Inhibition of growth was observed, but the magnitude of the effect was not reported. Autopsies that included histological examinations of the liver, lungs, kidneys, intestines and adrenals were conducted at the termination, but results were not reported specifically for pyrene-treated animals. Liver enlargement and fatty appearance were reported as general findings in "some" animals that were treated with pyrene, benzpyrene or methylcholanthrene, but the incidence of these effects in the pyrene-treated rats was not indicated. Interpretation of the results

of this study is complicated by the small group size, lack of control data, unreported number of animals, unreported magnitude of depressed weight gain and food consumption data and unreported incidence of hepatic alterations.

Holland et al. (1980) administered uncharacterized oil shale containing PAHs, including pyrene, to Syrian golden hamsters by inhalation of 50 mg respirable shale dust/m^3 for 4 hours/day, 4 days/week. The authors reported interim results indicating that shale dust caused little pulmonary epithelial or fibrotic reaction, but that retorted shales caused inflammation accompanied by fibrosis. Because of the uncharacterized nature of the test material, it is not possible to quantify these data for use in risk assessment.

5.6. OTHER RELEVANT INFORMATION

Four-day and 7-day single dose intraperitoneal LD_{50}s of 678 and 514 mg/kg, respectively, were determined for pyrene with mice (Salamone et al., 1981).

A single intraperitoneal injection of 150 mg/kg pyrene dissolved in DMSO produced minimal gross swelling and congestion in the livers of 6 male Sprague-Dawley rats after 24 and 72 hours (Yoshikawa et al., 1985). Gross pathologic effects in other unspecified tissues were not indicated. Small but significant increases in serum AST and bilirubin levels occurred 24 hours but not 72 hours after treatment, and significant changes in serum ALT, GGPT, LDH, glucose, BUN and creatine did not occur.

Pyrene in beeswax pellets was implanted in rat tracheas that had been implanted under the dorsal skin of isogenic recipients (Topping et al., 1978). Alterations in the respiratory mucosa developed during an 8-week observation period, including goblet-cell and transitional hyperplasia.

Intraperitoneal injection of 20 mg/kg pyrene to CFLP mice on day 18 of pregnancy did not cause any changes in the activities of lung pyruvate kinase or lactate dehydrogenase in the offspring when examined on gestation day 18 or 19 or postpartum days 1, 2, 3, 5, 7, 14, 28 or 35 (Rady et al., 1982).

5.7. SUMMARY

Intratracheal instillation of 3 mg pyrene suspended in saline at weekly intervals for 30 weeks did not produce tumors in the respiratory system (histologic examination) or other tissues (gross examination) in hamsters observed for life (Sellakumar and Shubik, 1974). Skin tumors were not induced in mice by twice or three-times weekly dermal application of pyrene for 1-2 years (Barry et al., 1935; Badger et al., 1940; Wynder and Hoffmann, 1959; Roe and Grant, 1964; Horton and Christian, 1974; Van Duuren and Goldschmidt, 1976). Mouse skin initiation-promotion studies involving initiation with benzo[a]pyrene (Roe and Grant, 1964), promotion with croton oil (Salaman and Roe, 1956) or promotion with TPA (Scribner, 1973; Wood et al., 1980) were negative or inconclusive (Scribner, 1973). Enhancement of the dermal carcinogenicity of benzo[a]pyrene in mice by simultaneous application of pyrene has, however, been reported (Goldschmidt et al., 1973; Van Duuren and Goldschmidt, 1976), indicating possible cocarcinogenicity of pyrene. Two subcutaneous injections of 10 mg pyrene crystals 4 months apart did not produce local tumors in mice after 12-18 months (Shear and Leiter, 1941).

Pyrene has been tested extensively in mutagenicity and other short-term assays. Pyrene was mutagenic in some tests with S. typhimurium in the presence of metabolic activation preparation (Kaden et al., 1979; Bridges et al., 1981; Matijasevic and Zeiger, 1985; Sakai et al., 1985) and produced

unscheduled DNA synthesis in cultured primary hamster hepatocytes (McQueen et al., 1983) and human fibroblasts (Robinson and Mitchell, 1981), mutation in mouse lymphoma L5178Y cells in vitro (Jotz and Mitchell, 1981), sister chromatid exchanges in CHO (Evans and Mitchell, 1981) and V79 (Popescu et al., 1977) cells in vitro and chromosomal aberrations in Chinese hamster V79 cells in vitro (Popescu et al., 1977). Genotoxicity of pyrene was not demonstrated in other assays using the same or different indicator organisms or endpoints, including morphological transformation in a variety of rat, mouse, hamster and guinea pig systems.

Pertinent data regarding teratogenicity or other reproductive effects of pyrene could not be located in the available literature as cited in the Appendix.

Inhibition of growth and enlarged, fatty livers were reported as effects in a group of six male rats maintained on diets that contained 2000 ppm pyrene for as long as 100 days (White and White, 1939). The significance of these findings is uncertain because incidences were not reported, a small number of animals was tested, the treatment durations were variable and unspecified, and control data were not reported. Pertinent data regarding toxic effects of chronic oral or inhalation exposure to pyrene could not be located in the available literature as cited in the Appendix.

A single intraperitoneal injection of 150 mg/kg pyrene produced minimal gross swelling and congestion of the liver and increased serum AST and bilirubin in rats (Yoshikawa et al., 1985).

6. AQUATIC TOXICITY

6.1. ACUTE

The available data concerning acute toxicity of pyrene to aquatic organisms are presented in Table 6-1. Of the six species for which there are data, the three arthropods are the most sensitive, with LC_{50} values of 4, 8 and 20 µg/ℓ for Daphnia magna, Artemia salina and Aedes aegypti, respectively (Kagan et al., 1985).

There is a wide discrepancy between the Daphnia magna LC_{50} value of 4 mg/ℓ (Kagan et al., 1985) and that reported by Bobra et al. (1983), 1820 µg/ℓ. This may be due to differences in lighting conditions used in the two studies. Like some other PAH (e.g., anthracene and fluoranthene), the toxicity of pyrene to aquatic organisms is greatly enhanced under natural sunlight or UV light. Pyrene was nontoxic to the five species tested by Kagan et al. (1985) at 1000 µg/ℓ in the dark; however, under light sources emitting primarily long-wavelength UV, the LC_{50} values in Table 6-1 were obtained.

6.2. CHRONIC

Pertinent data regarding chronic toxicity of pyrene to aquatic organisms could not be located in the available literature as cited in the Appendix.

6.3. PLANTS

The only data concerning effects of pyrene on aquatic plants were provided by Hutchinson et al. (1980), who reported EC_{50} values of 202 and 332 µg/ℓ for inhibition of photosynthesis in Chlamydomonas angulosa and Chlorella vulgaris, respectively.

TABLE 6-1

Acute Toxicity of Pyrene to Aquatic Organisms

Species	Concentration (µg/ℓ)	Effect	Reference
FISH			
Fathead minnow (*Pimephales promelas*)	220	24-hour LC_{50}	Kagan et al., 1985
Sea lamprey (*Petromyzon marinus*)	5000	nontoxic, 24 hours	Applegate et al., 1957
AMPHIBIANS			
Frog (*Rana pipiens*)	140	24-hour LC_{50}	Kagan et al., 1985
CRUSTACEANS			
Water flea (*Daphnia magna*)	4	24-hour LC_{50}	Kagan et al., 1985
	1820	48-hour LC_{50}	Bobra et al., 1983
Brine shrimp (*Artemia salina*)	8	24-hour LC_{50}	Kagan et al., 1985
INSECTS			
Mosquito (*Aedes aegypti*)	20	24-hour LC_{50}	Kagan et al., 1985

6.4. RESIDUES

Bioconcentration data for five species are presented in Table 6-2. The algae had the highest BCF values, 13,072 and 36,300 (Casserly et al., 1983; Davis et al., 1983), and rainbow trout the lowest, 72 (Gerhart and Carlson, 1978). Eadie et al. (1983) noted that sediment can be the principal source for PAH uptake by benthes such as the amphipod, Pontoporeia hoyi.

Monitoring data for pyrene residues in aquatic organisms are presented in Table 6-3. In general, the highest body burdens were reported for molluscs from polluted areas in Norway (Knutzen and Sortland, 1982) and the central Gulf of Mexico (Nulton and Johnson, 1981).

6.5. SUMMARY

Of six species tested, three arthropods were more sensitive to pyrene than fish or amphibians. LC_{50} values of 4, 8 and 20 µg/ℓ were reported for Daphnia magna, Artemia salina and Aedes aegypti, respectively. Like some other PAH, the toxicity of pyrene is greatly enhanced by UV light or sunlight (Kagan et al., 1985). Two algae species were less sensitive to pyrene than the three arthropods, with EC_{50} values of 202 and 332 µg/ℓ for inhibition of photosynthesis (Hutchinson et al., 1980). Reported BCF values ranged from 72 for rainbow trout (Gerhart and Carlson, 1978) to 36,300 for the alga, Selenastrum capricornutum (Casserly et al., 1983).

TABLE 6-2

Bioconcentration Data for Pyrene in Aquatic Organisms

Species	Concentration (µg/ℓ)	BCF	Remarks	Reference
FISH				
Goldfish (*Carassius auratus*)	NR	457	No comment	Ogata et al., 1984
Rainbow trout (*Salmo gairdneri*)	3.89	72	21-day BCF	Gerhart and Carlson, 1978
CRUSTACEANS				
Water flea (*Daphnia pulex*)	NR	2702	24-hour BCF	Southworth et al., 1976
MOLLUSCS				
Clam (*Tapes japonica*)	0.12-0.24	1400-2400	1-7 day BCF	Obana et al., 1983
PLANTS				
Algae (several species)	NR	13,072	natural pond community, 40-day exposure	Davis et al., 1983
Green alga (*Selenastrum capricornutum*)	NR	36,300	24-hour BCF	Casserly et al., 1983

NR = Not reported

TABLE 6-3

Monitoring Data for Pyrene Residues in Aquatic Organisms

Species	Location	Tissue	Tissue Concentration (ng/g)	Reference
FISH				
Eel, Anguilla rostrata	Lake Ontario	whole body	1.9	Lawrence and Weber, 1984
Lake trout, Salvelinus namaycush	Lake Maskinonge, Ontario	edible portions	<0.3	Pancirov and Brown, 1977
Smelt, Osmerus sp.	Lake Ontario	whole body	1.0-1.4	Lawrence and Weber, 1984
Cod, Gadus sp.	Tom's River, NJ	edible portions	<0.5	Pancirov and Brown, 1977
White sucker, Catostomus commersoni	eastern Lake Erie	stomach contents	2-74	Maccubbin et al., 1985
Catfish, Ictalurus sp.	Lake Ontario	whole body	1.8	Lawrence and Weber, 1984
White perch, Morone americana	Lake Ontario	whole body	2.3	Lawrence and Weber, 1984
Crappie, Pomoxis sp.	Lake Ontario	whole body	1.1	Lawrence and Weber, 1984
Menhaden, Brevoortia sp.	Raritan Bay	edible portions	<0.3	Pancirov and Brown, 1977
Sheepshead, Archasargus probatocephalus	central Gulf of Mexico	liver	10-60	Nulton and Johnson, 1981
		gill	20	
		flesh	30	
Red snapper, Lutjanus campenchanus	central Gulf of Mexico	gill	30	Nulton and Johnson, 1981
		flesh	20	
Spadefish, Chaetodipterus faber	central Gulf of Mexico	liver	20-50	Nulton and Johnson, 1981
		gill	10-40	
		flesh	80	
Creolefish, Paranthius furcifer	central Gulf of Mexico	liver	30	Nulton and Johnson, 1981
		gonad	10	
Amberjack, Seriola dumerili	central Gulf of Mexico	liver	10	Nulton and Johnson, 1981
Flounder (unspecified)	Atlantic Ocean, Long Island, NY	edible portions	2	Pancirov and Brown, 1977
English sole, Parophrys vetulus	Puget Sound, WA	stomach contents	93-2300	Malins et al., 1985b
Summer flounder, Paralichthys dentatus	Baltimore Canyon, Atlantic Ocean	whole body	2	Brown and Pancirov, 1979
Red lake, Arophysate chuss			<5	
Scup, Stenotomus chrysops			2	
Black sea bass, Centropritus stricta			<1	
Butterfish, Paprilus triacanthus			<1	

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TABLE 6-3 (cont.)

Species	Location	Tissue	Tissue Concentration (ng/g)	Reference
MOLLSUCS				
Sea scallop, *Placopeskin magellianicus*	Baltimore Canyon, Atlantic Ocean	whole body	4.1	Brown and Pancirov, 1979
	Central Gulf of Mexico	whole body	130	Hulton and Johnson, 1981
Paper scallop, *Amusium papyraceum*	Narragansett Bay, RI	whole body	34-92	Galloway et al., 1983
Mussel, *Mytilus edulis*	Norway, polluted areas	whole body	29-2054	Knutzen and Sortland, 1982
Mussel, *Mytilus edulis*	Falmouth, MA	whole body	2-4	Pancirov and Brown, 1977
Mussel *Mytilus edulis*	Norway, polluted areas	whole body	141-3128	Knutzen and Sortland, 1982
Limpet, *Patella vulgata*	Norway, polluted areas	whole body	32-204	Knutzen and Sortland, 1982
Periwinkle, *Littorina littorea*	central Gulf of Mexico	whole body	90	Hulton and Johnson, 1981
Eastern oyster, *Crassostrea virginica*	Long Island Sound	whole body	58	Pancirov and Brown, 1977
	Chincoteague, VA	whole body	0.5	Pancirov and Brown, 1977
	Chincoteague, VA	whole body	1	Pancirov and Brown, 1977
Clam (unspecified)	Darien, CN	whole body	12	Pancirov and Brown, 1977
	Linden, NJ	whole body	<3	Pancirov and Brown, 1977
CRUSTACEANS				
Brown shrimp, *Penaeus aztecus*	central Gulf of Mexico	whole body	30	Hulton and Johnson, 1981
Shrimp (unspecified)	Palacios, TX	edible portion	<0.3	Pancirov and Brown, 1977
Lobster, *Homarus americanus*	Atlantic Ocean, Eastern Canada	whole body	17.5	Dunn and Fee, 1979
	Chesapeake Bay	edible portion	<0.2	Pancirov and Brown, 1977
Crab (unspecified)	Raritan Bay	edible portion	6	Pancirov and Brown, 1977
MISCELLANEOUS INVERTEBRATES				
Polychaete worms (unspecified)	New York bight	whole body	3.8-200	Farrington et al., 1986
Starfish, *Asterias rubens*	Norway, polluted areas	whole body	10-18	Knutzen and Sortland, 1982
Sponge, *Halichondria panicea*	Norway, polluted areas	whole body	39	Knutzen and Sortland, 1982
PLANTS				
Bladder wrack, *Fucus vesiculosus*	Norway, polluted areas	whole body	28-286	Knutzen and Sortland, 1982
Toothed wrack, *Fucus serratus*	Norway, polluted areas	whole body	<4-224	Knutzen and Sortland, 1982
Knotted wrack, *Ascophyllum nodosum*	Norway, polluted areas	whole body	15-388	Knutzen and Sortland, 1982
Laminaria saccharina	Norway, polluted areas	whole body	36	Knutzen and Sortland, 1982
Ceramium rubrum	Norway, polluted areas	whole body	20	Knutzen and Sortland, 1982

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7. EXISTING GUIDELINES AND STANDARDS

7.1. HUMAN

Exposure criteria and TLVs have been developed for PAH as a class, as well as for several individual PAH. OSHA has set an 8-hour TWA concentration limit of 0.2 mg/m³ for the benzene-soluble fraction of coal tar pitch volatiles (anthracene, benzo[a]pyrene, phenanthrene, acridine, chrysene, pyrene) (OSHA, 1985). NIOSH (1977) recommends a concentration limit for coal tar, coal tar pitch, creosote and mixtures of these substances of 0.1 mg/m³ of the cyclohexane-extractable fraction of the sample, determined as a 10-hour TWA. NIOSH (1977) concluded that these specific coal tar products, as well as coke oven emissions, are carcinogenic and can increase the risk of lung and skin cancer in workers. NIOSH (1977) also recommends a ceiling limit for exposure to asphalt fumes of 5 mg airborne particulates/m³ of air.

Environmental quality criteria, which specify concentration limits intended to protect humans against adverse health effects, have been recommended for PAHs in ambient water. The U.S. EPA (1980) has recommended a concentration limit of 28 ng/ℓ for the sum of all carcinogenic PAHs in ambient water. This value is based on a mathematical extrapolation of the results from studies with mice treated orally with benzo[a]pyrene, and acknowledges the conservative assumption that all carcinogenic PAHs are equal in potency to benzo[a]pyrene. On the basis of the animal bioassay data, daily consumption of water containing 28 ng/ℓ of carcinogenic PAHs over an entire lifetime is estimated to keep the lifetime risk of cancer development below one chance in 100,000.

The EPA has not recommended an ambient water quality criterion for noncarcinogenic PAHs as a class. The U.S. EPA (1980) acknowledged that data suitable for quantitative risk assessment of noncarcinogenic PAHs are essentially nonexistent.

7.2. AQUATIC

Guidelines and standards for the protection of aquatic biota from the toxic effects of pyrene in particular could not be located in the available literature as cited in the Appendix. The U.S. EPA (1980) made the following conclusions: that acute toxicity of PAH in general to saltwater biota recurred at concentrations as low as 300 µg/ℓ and would be expected to occur at lower concentrations in species more sensitive than those tested; that acute toxicity of PAH in general to saltwater biota occurred at concentrations in species more sensitive than those tested; and that the pyrene data base at that time was inadequate to recommend criteria or draw conclusions about chronic toxicity or acute toxicity freshwater organisms.

8. RISK ASSESSMENT

Intratracheal instillation of 3 mg pyrene suspended in saline at weekly intervals for 30 weeks did not produce tumors in the respiratory system (histologic examination) or other tissues (gross examination) in hamsters observed for life (Sellakumar and Shubik, 1974). Skin tumors were not induced in mice by twice or three-times weekly dermal application of pyrene for 1-2 years (Barry et al., 1935; Badger et al., 1940; Wynder and Hoffmann, 1959; Roe and Grant, 1964; Horton and Christian, 1974; Van Duuren and Goldschmidt, 1976). Mouse skin initiation-promotion studies involving initiation with benzo[a]]pyrene (Roe and Grant, 1964), promotion with croton oil (Salaman and Roe, 1956) or promotion with TPA (Scribner, 1973; Wood et al., 1980) were negative or inconclusive (Scribner, 1973). Enhancement of the dermal carcinogenicity of benzo[a]pyrene in mice by simultaneous application of pyrene has, however, been reported (Goldschmidt et al., 1973; Van Duuren and Goldschmidt, 1976), indicating possible cocarcinogenicity of pyrene. Two subcutaneous injections of 10 mg pyrene crystals 4 months apart did not produce local tumors in mice after 12-18 months (Shear and Leiter, 1941).

Pyrene has been tested extensively in mutagenicity and other short-term assays. Pyrene was mutagenic in some tests with S. typhimurium in the presence of metabolic activation preparation (Kaden et al., 1979; Bridges et al., 1981; Matijasevic and Zeiger, 1985; Sakai et al., 1985) and produced unscheduled DNA synthesis in cultured primary hamster hepatocytes (McQueen et al., 1983) and human fibroblasts (Robinson and Mitchell, 1981), mutation in mouse lymphoma L5178Y cells in vitro (Jotz and Mitchell, 1981), sister chromatid exchanges in CHO (Evans and Mitchell, 1981) and V79 (Popescu et

al., 1977) cells *in vitro* and chromosomal aberrations in Chinese hamster V79 cells *in vitro* (Popescu et al., 1977). Genotoxicity of pyrene was not demonstrated in other assays using the same or different indicator organisms or endpoints, including morphological transformation in a variety of rat, mouse, hamster and guinea pig systems.

Pertinent data regarding teratogenicity or other reproductive effects of pyrene could not be located in the available literature as cited in the Appendix.

Inhibition of growth and enlarged, fatty livers were reported as effects in a group of six male rats maintained on diets that contained 2000 ppm pyrene for as long as 100 days (White and White, 1939). The significance of these findings is uncertain because incidences were not reported and control data were lacking. Information regarding toxic effects of chronic oral or inhalation exposure to pyrene could not be located in the available literature as cited in the Appendix.

A single intraperitoneal injection of 150 mg/kg pyrene produced minimal gross swelling and congestion of the liver and increased serum AST and bilirubin in rats (Yoshikawa et al., 1985).

As indicated above, pyrene was not tumorigenic when instilled into the trachea of hamsters or applied to the skin of mice. Mouse skin initiation promotion studies were negative or inconclusive. Although pyrene does not appear to be active as a complete carcinogen or initiator or promoter in mouse skin studies, cocarcinogenicity with benzo[a]pyrene has been reported in dermal studies with mice. The subcutaneous injection study with mice is inadequate for evaluation of carcinogenicity because of the limited exposure schedule. Although genotoxicity of pyrene has been demonstrated in several short-term assays, the available data do not provide evidence that pyrene is a procarcinogen.

Calculation of an RfD based on the growth inhibition and fatty livers in rats (White and White, 1939) is precluded by limitation of data quality as detailed previously, particularly the small number of animals, lack of control data, unreported incidence and magnitude of effects. These data were not evaluated in the U.S. EPA (1984) Health Effects Assessment of pyrene, which also did not calculate an RfD because of the lack of data.

9. REPORTABLE QUANTITIES

9.1. REPORTABLE QUANTITY (RQ) RANKING BASED ON CHRONIC TOXICITY

Inhibition of growth and enlarged, fatty livers were reported as effects in a group of six male rats of unspecified strain that were maintained on diets containing 2000 ppm pyrene for ≤100 days (White and White, 1939). The significance of these effects is difficult to ascertain because of deficiencies in experimental design and inadequate reporting. These include the small group size and lack of an unexposed control group. In addition, the magnitude of the growth inhibition was not indicated and food consumption data were not reported. The hepatic effects were reported as general findings in "some" animals that were treated with pyrene, benzpyrene or methylcholanthrene, but the incidence of these effects in the pyrene-treated rats was not indicated.

Holland et al. (1980) administered uncharacterized oil shale containing PAH, including pyrene, to Syrian golden hamsters by inhalation of 50 mg respirable shale dust/m^3 for 4 hours/day, 4 days/week. The authors reported interim results indicating that shale dust caused little pulmonary epithelial or fibrotic reaction, but that retorted shales caused inflammation accompanied by fibrosis.

Weaver and Gibson (1979) exposed pregnant rats to graded airborne concentrations of uncharacterized oil shale containing PAH, including pyrene, on days 6-15 of gestation. No treatment-related teratogenic effects were observed during examination of fetuses obtained by Caesarean section on day 20 of gestation.

Calculation of an RQ for pyrene based on the growth inhibition or fatty livers in rats (White and White, 1939) is precluded by the limitations of data quality as indicated above. Because of the uncharacterized nature of

the test materials in the inhalation studies with hamsters (Holland et al., 1980) and rats (Weaver and Gibson, 1979), it is not possible to quantify these data for use in deriving an RQ (Table 9-1) to reflect hazard associated with exposure to pyrene (U.S. EPA, 1983).

9.2. WEIGHT OF EVIDENCE AND POTENCY FACTOR ($F=1/ED_{10}$) FOR CARCINOGENICITY

Intratracheal instillation of 3 mg pyrene suspended in saline at weekly intervals for 30 weeks did not produce tumors in the respiratory system (histologic examination) or other tissues (gross examination) in hamsters observed for life (Sellakumar and Shubik, 1974). Skin tumors were not induced in mice by twice or three-times weekly dermal application of pyrene for 1-2 years (Barry et al., 1935; Badger et al., 1940; Wynder and Hoffmann, 1959; Roe and Grant, 1964; Horton and Christian, 1974; Van Duuren and Goldschmidt, 1976). Mouse skin initiation-promotion studies involving initiation with benzo[a]pyrene (Roe and Grant, 1964), promotion with croton oil (Salaman and Roe, 1956) or promotion with TPA (Scribner, 1973; Wood et al., 1980) were negative or inconclusive (Scribner, 1973). Enhancement of the dermal carcinogenicity of benzo[a]pyrene in mice by simultaneous application of pyrene has, however, been reported (Goldschmidt et al., 1973; Van Duuren and Goldschmidt, 1976), indicating possible cocarcinogenicity of pyrene. Two subcutaneous injections of 10 mg pyrene crystals 4 months apart did not produce local tumors in mice after 12-18 months (Shear and Leiter, 1941).

Pyrene has been tested extensively in mutagenicity and other short-term assays. Pyrene was mutagenic in some tests with S. typhimurium in the presence of metabolic activation preparation (Kaden et al., 1979; Bridges et al., 1981; Matijasevic and Zeiger, 1985; Sakai et al., 1985) and produced unscheduled DNA synthesis in cultured primary hamster hepatocytes (McQueen

TABLE 9-1

Pyrene
Minimum Effective Dose (MED) and Reportable Quantity (RQ)

Route:	
Dose:	
Effect:	
Reference:	
RV_d:	
RV_e:	
Composite Score:	
RQ:	Data are not sufficient to derive an RQ.

et al., 1983) and human fibroblasts (Robinson and Mitchell, 1981), mutation in mouse lymphoma L5178Y cells in vitro (Jotz and Mitchell, 1981), sister chromatid exchanges in CHO (Evans and Mitchell, 1981) and V79 (Popescu et al., 1977) cells in vitro and chromosomal aberrations in Chinese hamster V79 cells in vitro (Popescu et al., 1977). Genotoxicity of pyrene was not demonstrated in other assays using the same or different indicator organisms or endpoints, including carcinogenic transformation in a variety of rat, mouse, hamster and guinea pig systems.

Although cocarcinogenicity of pyrene with benzo[a]pyrene has been demonstrated in skin application studies with mice and short-term assays provide limited evidence of genotoxicity, the available data do not indicate that pyrene is a procarcinogen. Carcinogenicity testing of pyrene by the oral or inhalation routes, however, has not been conducted.

IARC (1983) reported that there was insufficient evidence regarding the carcinogenic risk to humans and experimental animals associated with oral or inhalation exposure to pyrene. Applying the criteria for evaluation of the overall weight of evidence for the carcinogenic potential for humans proposed by the U.S. EPA (1986b), pyrene is most appropriately designated a Group D - Not Classified chemical. Direct hazard ranking of pyrene under CERCLA is therefore not possible.

10. REFERENCES

Applegate, V.C., J.H. Howell, A.E. Hall, Jr. and M.A. Smith. 1957. Toxicity of 4346 chemicals to larval lampreys and fishes. Spec. Sci. Rep. Fish. No. 207, Fish. Wildl. Serv., USDI, Washington, DC. 157 p.

Badger, G.M., J.W. Cook, C.L. Hewett, et al. 1940. The production of cancer by pure hydrocarbons. V. Proc. R. Soc. London Ser. B. 129: 439-467.

Barry, G., I.A. Cook, G. Haslewood, C.I. Hewett and E.L. Kennaway. 1935. The production of cancer by pure hydrocarbons. Part III. Proc. Roy. Soc. (London). 117: 318-351.

Behymer, T.D. and R.A. Hites. 1985. Photolysis of polyaromatic hydrocarbons adsorbed on simulated atmospheric particles. Environ. Sci. Technol. 19: 1004-1006.

Benoit, F.M., G.L. Lebel and D.T. Williams. 1979. The determination of polycyclic aromatic hydrocarbons at the ng/ℓ level in Ottawa tap water. Int. J. Environ. Anal. Chem. 6: 277-287.

Bobra, A.M., W.Y. Shiu and D. Mackay. 1983. A predictive correlation for the acute toxicity of hydrocarbons and chlorinated hydrocarbons to the water flea (Daphnia magna). Chemosphere. 12(9-10): 1121-1130.

Boehm, P.D. and J.W. Farrington. 1984. Aspects of the polycyclic aromatic hydrocarbon geochemistry of recent sediments in the Georges bank region. Environ. Sci. Technol. 18: 840-845.

Boyland, E. and P. Sims. 1964. Metabolism of polycyclic compounds. The metabolism of pyrene in rats and rabbits. Biochem. J. 90: 391-398.

Bridges, B.A., E. Zeiger and D.B. McGregor. 1981. Summary report on the performance of bacterial mutation assays. In: Evaluation of Short-Term Tests for Carcinogens. Report of the International Collaborative Program. Progress in Mutation Research, F.J. deSerres and J. Ashby, Ed., Vol. 1. Elsevier/North Holland, NY. p. 49-67.

Brown, R.A. and R.J. Pancirov. 1979. Polynuclear aromatic hydrocarbons in Baltimore Canyon fish. Environ. Sci. Technol. 13(7): 878-879.

Butkovic, V., L. Klasinc, M. Orhnovic, J. Turk and H. Gusten. 1983. Reaction rates of polynuclear aromatic hydrocarbons with ozone in water. Environ. Sci. Technol. 17: 546-548.

Carlson, R.M., A.R. Oyler, E.H. Gerhart, et al. 1979. Implications to the aquatic environment of polynuclear aromatic hydrocarbons liberated from northern Great Plains coal. U.S. EPA, Duluth, MN. EPA 600/3-79-093.

Casserly, D.M., E.M. Davis, T.D. Downs and R.K. Guthrie. 1983. Sorption of organics by Selenastrum capricornutum. Water Res. 17(11): 1591-1594.

Cole, R.H., R.E. Frederick, R.P. Healy and R.G. Rolan. 1984. Preliminary findings of the priority pollutant monitoring project of the nationwide urban runoff program. J. Water Pollut. Control Fed. 56: 898-908.

Darroudi, F. and A.J. Natarajan. 1985. Cytostatic drug activity in plasma, a bioassay for detecting mutagenicity of directly and indirectly acting chemicals, an evaluation of 20 chemicals. Mutat. Res. 143(4): 263-269.

Davis, E.M., J.E. Turley, D.M. Casserly and R.K. Guthrie. 1983. Partitioning of selected organic pollutants in aquatic ecosystems. In: Biodeterioration 5, T.A. Oxley and S. Barry, Ed. John Wiley and Sons, New York, NY. p. 176-184.

Dean, B.J. 1981. Activity of 27 coded compounds in the RL, chromosome assay. In: Evaluation of Short-Term Tests for Carcinogens. Report of the International Collaborative Program. Progress in Mutation Research, F.J. deSerres and J. Ashby, Ed., Vol. 1. Elsevier/North Holland, NY. p. 570-579.

Dennis, M.J., R.C Massey, D.J. McWeeney, M.E. Knowles and D. Watson. 1983. Analysis of polycyclic aromatic hydrocarbons in UK total diets. Food Chem. Toxicol. 21: 569-574.

Dunn, B.P. and J. Fee. 1979. Polycyclic aromatic hydrocarbon carcinogens in commercial seafoods. J. Fish. Res. Board Can. 36(12): 1469-1476.

Eadie, B.J., W. Faust, W.S. Gardner and T. Nalepa. 1982. Polycyclic aromatic hydrocarbons in sediments and associated benthos in Lake Erie. Chemosphere. 11: 185-191.

Eadie, B.J., W.R. Faust, P.F. Landrum, N.R. Morehead, W.S. Gardner and T. Nalepa. 1983. Bioconcentrations of PAH by some benthic organisms of the Great Lakes. Polynucl. Aromat. Hydrocarbons, Int. Symp., M. Cooke, A.J. Dennis, Ed., Battelle Press, CO. p. 437-449. [CA 99(25):207586k]

Eiceman, G.A., R.E. Clement and F.W. Karasek. 1981. Variations in concentrations of organic compounds including PCDDS and polynuclear aromatic hydrocarbons in fly ash from a municipal incinerator. Anal. Chem. 53: 955-959.

Elder, V.A., B.L. Proctor and R.A. Hites. 1981. Organic compounds found near dump sites in Niagara Falls, NY. Environ. Sci. Technol. 15: 1237-1243.

Evans, E.L. and A.D. Mitchell. 1981. Effect of 20 coded chemicals on sister chromatid exchange frequencies in cultured Chinese hamster cells. In: Evaluation of Short-Term Tests for Carcinogens. Report of the International Collaborative Program. Progress in Mutation Research, F.J. deSerres and J. Ashby, Ed., Vol. 1. Elsevier/North Holland, NY. p. 538-550.

Farrington, J.W., S.G. Wakeham, J.B. Livramento, B.W. Tripp and J.M. Teal. 1986. Aromatic hydrocarbons in New York Bight polychaetes: Ultraviolet fluorescence analyses and gas chromatography-mass spectrometry analyses. Environ. Sci. Technol. 20(1): 69-72.

Fazio, T. and J.W. Howard. 1983. Polycyclic aromatic hydrocarbons in foods. In: Handbook of Polycyclic Aromatic Hydrocarbons, A. Bjoerseth, Ed. Marcel Dekker, Inc., New York, NY. p. 461-505.

Galloway, W.B., J.L. Lake, D.K. Phelps, et al. 1983. The mussel watch: Intercomparison of trace level constituent determinations. Environ. Toxicol. Chem. 2(4): 395-410.

Gerhart, E.H. and R.M. Carlson. 1978. Hepatic mixed-function oxidase activity in rainbow trout exposed to several polycyclic aromatic compounds. Environ. Res. 17(2): 284-295.

Goldschmidt, B.M., C. Katz and B.L. Van Duuren. 1973. The cocarcinogenic activity of non-carcinogenic aromatic hydrocarbons (Abstract No. 334). Proc. Am. Assoc. Cancer Res. 17: 84.

Goodley, P.C. and M. Gordon. 1976. Characterization of industrial organic compounds in water. Kentucky Academy of Science. 37: 11-15.

Great Lakes Water Quality Board. 1983. An inventory of chemical substances identified in the Great Lakes ecosystem. Vol. 1-summary. Report to the Great Lakes Water Quality Board. Windsor Ontario, Canada. p. 195.

Greenberg, A., F. Darack, R. Harkov, P. Lioy and J. Daisey. 1985. Polycyclic aromatic hydrocarbons in New Jersey. A comparison of winter and summer concentrations over a two-year period. Atmos. Environ. 19: 1325-1339.

Griest, W.H. 1980. Multicomponent polycyclic aromatic hydrocarbon analysis of inland water and sediment. In: Hydrocarbon Halo. Hydrocarbon Aquatic Environ., B.K. Afghan and D. Mackay, Ed. Plenum Press, New York, NY. p. 173-183.

Groenewegen, D. and H. Stolp. 1981. Microbial breakdown of polycyclic aromatic hydrocarbons. In: Decomposition of Toxic and Nontoxic Organic Compounds in Soil, M.R. Overcash, Ed. Ann Arbor Science Publ., Ann Arbor, MI. p. 223-240.

Hashimoto, Y., K. Tokura, H. Kishi and W.M.J. Strachan. 1984. Prediction of seawater solubility of aromatic compounds. Chemosphere. 13(8): 881-888.

Hawley, G.G. 1981. The Condensed Chemical Dictionary, 10th ed. Van Nostrand Reinhold Co., New York, NY. p. 872.

Heit, M. 1985. The relationship of a coal fired power plant to the levels of polycyclic aromatic hydrocarbons (PAH) in the sediment of Cayuga Lake. Water Air Soil Pollut. 24: 41-61.

Hites, R.A. 1979. Sources and fates of industrial organic chemicals. A case study. Proc. Natl. Conf. Munic. Sludge Manage. 8: 107-119.

Ho, C.H., B.R. Clark, M.R. Guerin, B.D. Barkenbus, T.K. Bao and J.L. Epler. 1981. Analytical and biological analyses of test materials from the synthetic fuel technologies. IV. Studies of chemical structure-mutagenic activity relationships of aromatic nitrogen compounds relevant to synfuels. Mutat. Res. 85: 335-345.

Hoffman, E.J., G.L. Mills, J.S. Latimer and J.G. Quinn. 1984. Urban runoff as a source of polycyclic aromatic hydrocarbons to coastal waters. Environ. Sci. Technol. 18: 580-587.

Holland, L.M., W.D. Spall and L.L. Garcia. 1980. Inhalation toxicology of oil shale-related materials. Health Implic. New Energy Technol., W.N. Rom and V.E. Archer, Ed. Ann. Arbor Science Publishers, Inc., Ann Arbor, MI. p. 515-526.

Horton, A.W. and G.M. Christian. 1974. Cocarcinogenic versus incomplete carcinogenic activity among aromatic hydrocarbons. Contrast between chrysene and benzo[b]triphenylene. J. Natl. Cancer Inst. 53: 1017-1020.

Hoy, C.A., E.P. Salazar and L.H. Thompson. 1984. Rapid detection of DNA-damaging agents using repair-deficient CHO cells. Mutat. Res. 130(5): 321-332.

Hutchinson, T.C., J.A. Hellebust, D. Tam, D. Mackay, R.A. Mascarenhas and W.Y. Shiu. 1980. The correlation of the toxicity to algae of hydrocarbons and halogenated hydrocarbons with their physical-chemical properties. Environ. Sci. Res. 16: 577-586.

IARC (International Agency for Research on Cancer). 1983. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Pyrene. In: Polynuclear Aromatic Compounds, Part I. Chemical, Environmental and Experimental Data. WHO, IARC, Lyons, France. Vol. 32, p. 431-446.

Jacob, J., G. Grimmer, G. Raab and A. Schmoldt. 1982. The metabolism of pyrene by rat liver microsomes and the influence of various mono-oxygenase inducers. Xenobiotica. 12: 45-53.

Johnson, A.C., P.F. Larsen, D.F. Gadbois and A.W. Humason. 1985. The distribution of polycyclic aromatic hydrocarbons in the surficial sediments of penobscot bay (Maine, USA) in relation to possible sources and other sites world wide. Marine Environ. Res. 15: 1-16.

Jotz, M.M. and A.D. Mitchell. 1981. Effects of 20 coded chemicals on the forward mutation frequency at the thymidine kinase locus in L5178Y mouse lymphoma cells. In: Evaluation of Short-Term Tests for Carcinogens. Report of the International Collaborative Program. Progress in Mutation Research, F.J. deSerres and J. Ashby, Ed. Elsevier/North Holland, NY. Vol. 1, p. 580-593.

Jungclaus, G.A., L.M. Games and R.A. Hites. 1976. Identification of trace organic compounds in tire manufacturing plant wastewaters. Anal. Chem. 48: 1894-1896.

Jungclaus, G.A., V. Lopez-Avila and R.A. Hites. 1978. Organic compounds in an industrial waste water. A case study of their environmental impact. Environ. Sci. Technol. 12: 88-96.

Kaden, D.A., R.A. Hites and W.G. Thilly. 1979. Mutagenicity of soot and associated polycyclic aromatic hydrocarbons to Salmonella typhimurium. Cancer Res. 39: 4152-4159.

Kagan, J., E.D. Kagan, I.A. Kagan, P.A. Kagan and S. Quigley. 1985. The phototoxicity of noncarcinogenic polycyclic aromatic hydrocarbons in aquatic organisms. Chemosphere. 14(11-12): 1829-1834.

Karickhoff, S.W. 1981. Semi-epirical estimation of sorption of hydrophobic pollutants on natural sediments and soils. Chemosphere. 10: 833-846.

Karickhoff, S.W. and K.R. Morris. 1985. Sorption dynamics of hydrophobic pollutants in sediment suspension. Environ. Toxicol. Chem. 4: 469-479.

Karickhoff, S.W., D.S. Brown and T.A. Scott. 1979. Sorption of hydrophobic pollutants on natural sediments. Water Res. 13: 241-248.

Keimig, S.D., K.W. Kirby, D.P. Morgan, J.E. Keiser and T.D. Hubert. 1983. Identification of 1-hydroxypyrene as a major metabolite of pyrene in pig urine. Xenobiotica. 13(7): 415-420.

Keller, C.D. and T.F. Bidleman. 1984. Collection of airborne polycyclic aromatic hydrocarbons and other organics with a glass fiber filter-polyurethane foam system. Atmos. Environ. 18: 837-845.

Kenaga, E.E. 1980. Predicted bioconcentration factors and soil sorption coefficients of pesticides and other chemicals. Ecotoxicol. Environ. Safety. 4: 26-38.

Kerkhoff, M.J., T.M. Lee, E.R. Allen, D.A. Lundgren and J.D. Winefordner. 1985. Spectral fingerprinting of polycyclic aromatic hydrocarbons in high-volume ambient air samples by constant energy synchronous luminescence spectroscopy. Environ. Sci. Technol. 19: 695-699.

Knutzen, J. and B. Sortland. 1982. Polycyclic aromatic hydrocarbons (PAH) in some algae and invertebrates from moderately polluted parts of the coast of Norway. Water Res. 16(4): 421-428.

Kuntz, K.W. 1984. Toxic contaminants in the Niagara River. 1975-1982. Technical Bulletin No. 134. Burlington, Ontario.

Kveseth, K., B. Sortland and T. Bonkn. 1982. Polycyclic aromatic hydrocarbons in sewage, mussels and tap water. Chemosphere. 11: 623-639.

Larsen, P.F., D.F. Gadbois, A.C. Johnson and L.F. Doggett. 1983. Distribution of polycyclic aromatic hydrocarbons in the surficial sediments of Casco Bay, Maine. Bull. Environ. Contam. Toxicol. 30: 530-535.

LaVoie, E., V. Bedenko, N. Hirota, S.S. Hecht and D. Hoffmann. 1979. A comparison of the mutagenicity, tumor-initiating activity and complete carcinogenicity of polynuclear aromatic hydrocarbons. In: Polynuclear Aromatic Hydrocarbons, P.W. Jones and P. Leber, Ed. Ann Arbor Science Publishers, Ann Arbor, MI. p. 705-721.

Lawrence, J.F. and D.F. Weber. 1984. Determination of polycyclic aromatic hydrocarbons in Canadian samples of processed vegetables and dairy products by liquid chromatography with fluorescence detection. J. Agric. Food Chem. 32: 794-797.

Ligocki, M.P., C. Leuenberger and J.F. Pankow. 1985a. Trace organic compounds in rain-II. Gas scavenging of neutral organic compounds. Atmos. Environ. 19: 1609-1617.

Ligocki, M.P., C. Leuenberger, and J.F. Pankow. 1985b. Trace organic compounds in rain-III. Particle scavenging of neutral organic compounds. Atmos Environ. 19: 1619-1626.

Lunde, G. and A. Bjoerseth. 1977. Polycyclic aromatic hydrocarbons in long-range transported aerosols. Nature. 268: 518-519.

Lyman, W.J., W.F. Reehl and D.H. Rosenblatt. 1982. Handbook of Chemical Property Estimation Methods. McGraw-Hill Book Co., New York. p. 15-16 to 15-29.

Mabey, W.R., J.H. Smith, T.R. Podoll, et al. 1981. Aquatic Fate Process Data for Organic Priority Pollutants. U.S. EPA, Monitoring and Data Support Division (WH 553), Office of Water Regulations and Standards, Washington, DC. EPA 440/4-82-014.

Maccubbin, A.E., P. Black, L. Trzeciak and J.J. Black. 1985. Evidence of polynuclear aromatic hydrocarbons in the diet of bottom-feeding fish. Bull. Environ. Contam. Toxicol. 34(6): 876-882.

Mackay, D. 1982. Correlation of bioconcentration factors. Environ. Sci. Technol. 16: 274-278.

Mackay, D., W.Y. Shiu, A. Bobra, et al. 1982. Volatilization of Organic Pollutants from Water. U.S. EPA, Athens, GA. EPA 600/53-82-019. NTIS PB82-230-939.

Malins, D.C., M.M. Krahn, M.S. Myers, et al. 1985a. Toxic chemicals in sediments and biota from a creosote-polluted harbor. Relationships with hepatic neoplasms and other hepatic lesions in English sole (Parophrys vetulus). Carcinogenesis. 6: 1463-1469.

Malins, D.C., M.M. Krahn, D.W. Brown, et al. 1985b. Toxic chemicals in marine sediment and biota from Mukiteo, Washington. Relationships with hepatic neoplasms and other hepatic lesions in English sole (Parophrys vetulus). J. Natl. Cancer Inst. 74(2): 487-494.

Mamber, S.W., V. Bryson and S.E. Katz. 1983. The Escherichia coli WP2/WP100 rec assay for detection of potential chemical carcinogens. Mutat. Res. 119(2): 135-144.

Mamber, S.W., V. Bryson and S.E. Katz. 1984. Evaluation of the Escherichia coli K12 inductest for detection of potential chemical carcinogens. Mutat. Res. 130(3): 141-152.

Matijasevic, Z. and E. Zeiger. 1985. Mutagenicity of pyrene in Salmonella. Mutat. Res. 142(4): 149-152.

Matsumoto, H. and T. Kashimoto. 1985. Average daily respiratory intake of polycyclic aromatic hydrocarbons in ambient air determined by capillary gas chromatography. Bull. Environ. Contam. Toxicol. 34: 17-23.

McCann, J., E. Choi, E. Yamasaki and B.N. Ames. 1975. Detection of carcinogens as mutagens in the Salmonella/microsome test. Assay of 300 chemicals. Proc. Natl. Acad. Sci. USA. 72: 5135-5139.

McFall, J.A., W.Y. Huang and J.L. Laseter. 1979. Organics at the air-water interface of Lake Pontchartrain. Bull. Environ. Contam. Toxicol. 22: 80-87.

McQueen, C.A., D.M. Kreiser and G.M. Williams. 1983. The hepatocyte primary culture/DNA repair assay using mouse or hamster hepatocytes. Environ. Mutagen. 5: 1-8.

Merrill, E.G. and T.L. Wade. 1985. Carbonized coal products as a source of aromatic hydrocarbons to sediments from a higher industrial estuary. Environ. Sci. Technol. 19: 597-603.

Miller, M.M., S.P. Wasik, G.L. Huang, W-Y Shiu and D. Mackay. 1985. Relationships between octanol-water partition coefficient and aqueous solubility. Environ. Sci. Technol. 19: 522-529.

Mitchell, C.E. and K. Tu. 1977. Distribution, retention and elimination of pyrene in rats following inhalation. Annu. Rep. Inhalation Toxicol. Res. Inst. p. 350-355.

Mitchell, C.E. and K.W. Tu. 1979. Distribution, retention and elimination of pyrene in rats after inhalation. J. Toxicol. Environ. Health. 5: 1171-1179.

NAS (National Academy of Sciences). 1972. Biologic Effects of Atmospheric Pollutants. Particulate Polycyclic Organic Matter. NAS, Washington, DC. p. 63-81.

Nielsen, J.B. and O. Anderson. 1986. The peritoneal cell carcinogenicity test. Mutat. Res. 173(2): 153-156.

Nikonova, T.V. 1977. Transplacental action of benzo[a]pyrene and pyrene. Bull. Exp. Biol. Med. 84: 1025-1027.

NIOSH (National Institute for Occupational Safety and Health). 1977. Criteria for a Recommended Standard...Occupational Exposure to Coal Tar Products, U.S. DHEW, PHS, CDC, Rockville, MD. Publ. No. 78-107.

NRC (National Research Council). 1983. Polycyclic Aromatic Hydrocarbons: Evaluation of Sources and Effects. Natl. Acad. Press, Washington, DC.

Nulton, C.P. and D.E. Johnson. 1981. Aromatic hydrocarbons in marine tissues from the Central Gulf of Mexico. J. Environ. Sci. Health. Part A. A16(3): 271-288.

Obana, H., S. Hori, A. Nakamura and T. Kashimoto. 1983. Uptake and release of polynuclear aromatic hydrocarbons by short-necked clams (Tapes japonica). Water Res. 17(9): 1183-1188.

Ogata, M., K. Fujisawa, Y. Ogino and E. Mano. 1984. Partition coefficients as a measure of bioconcentration potential of crude oil compounds in fish and shellfish. Bull. Environ. Contam. Toxicol. 33: 561-567.

Ohio River Valley Water Sanitation Commission. 1979. Water treatment process modifications for trihalomethane control and organic substances in the Ohio river. Prepared in fulfillment of U.S. EPA Grant R-804615 for project activities 10/76-8/79. Cincinnati, OH.

OSHA (Occupational Safety and Health Administration). 1985. OSHA Safety and Health Standards. 29 CFR 1910.1000.

Overton, E.B., S.W. Mascarella, J.A. McFall and J.L. Laseter. 1980. Organics in the water column and air-water interface samples of Mississippi River water. Chemosphere. 9: 629-633.

Pancirov, R.J. and R.A. Brown. 1977. Polynuclear aromatic hydrocarbons in marine tissues. Environ. Sci. Technol. 11(10): 989-992.

Pearlman, R.S., S.H. Yalkowsky and S. Banerjee. 1984. Water solubilities of polynuclear aromatic and heteroaromatic compounds. J. Phys. Chem. Ref. Data. 13: 555-562.

Popescu, N.C., D. Turnbull and J.A. DiPaolo. 1977. Sister chromatid exchange and chromosome aberration analysis with the use of several carcinogens and noncarcinogens. Brief communication. J. Natl. Cancer Inst. 59: 289-293.

Prahl, F.G., E. Crecellus and R. Carpenter. 1984. Polycyclic aromatic hydrocarbons in Washington coastal sediments. An evaluation of atmospheric and riverene roots of introduction. Environ. Sci. Technol. 18: 687-693.

Quillardet, P., C. DeBellecombe and M. Hofnung. 1985. The SOS Chromotest, a colorimetric bacterial assay for genotoxins. Validation study with 83 compounds. Mutat. Res. 147(3): 79-95.

Rady, P., I. Arany, E. Uzvolgyi, F. Bojan and P. Kertai. 1982. Transplacental effects of carcinogens and noncarcinogens on activities of pyruvate kinase and lactate dehydrogenase as well as isozymic pattern of LDH in mouse lung. Toxicology. 24(3-4): 251-257.

Readman, J.W., R.F.C. Mantoura, M.M. Rhead and L. Brown. 1982. Aquatic distribution and heterotrophic degradation of polycyclic aromatic hydrocarbons (PAH) in the Tamar Estuary. Estuarine, Coastal. Shelf. Sci. 14: 369-389.

Robinson, D.E. and A.D. Mitchell. 1981. Unscheduled DNA synthesis response of human fibroblasts, WI-38 cells, to 20 coded chemicals. In: Evaluation of Short-Term Tests for Carcinogens. Report of the International Collaborative Program. Progress in Mutation Research, F.J. deSerres and J. Ashby, Ed. Elsevier/North Holland, NY. Vol. 1, p. 517-527.

Roe, F.J.C. and G.A. Grant. 1964. Tests of pyrene and phenanthrene for incomplete carcinogenic and anticarcinogenic activity (Abstract). Br. Emp. Cancer Campaign. 41: 59-60. (Cited in IARC, 1983)

Rostad, C.E., W.E. Pereira and M.F. Hult. 1985. Partitioning studies of coal-tar constituents in a two-phase contaminated groundwater system. Chemosphere. 14: 1023-1036.

Ruepert, C., A. Grinusis and H. Govers. 1985. Prediction of partition coefficients of unsubstituted polycyclic aromatic hydrocarbons from C_{18} chromatographic and structural properties. Chemosphere. 14: 279-291.

Sakai, M., D. Yoshida and S. Mizusaki. 1985. Mutagenicity of polycyclic aromatic hydrocarbons and quinones on Salmonella typhimurium TA97. Mutat. Res. 156(1-2): 61-67.

Salaman, M.H. and F.J.C. Roe. 1956. Further tests for tumour-initiating activity. N,N-Di(2-chloroethyl)-p-aminophenylbutyric acid (CB1348) as an initiator of skin tumour formation in the mouse. Br. J. Cancer. 10: 363-378.

Salamone, M.F., J.A. Heddle and M. Katz. 1981. Mutagenic activity of 41 compounds in the in vivo micronucleus assay. In: Evaluation of Short-Term Tests for Carcinogens. Report of the International Collaborative Program. Progress in Mutation Research, F.J. deSerres and J. Ashby, Ed. Elsevier/ North Holland, NY. Vol. 1, p. 686-697.

Santodonato, J., P. Howard and D. Basu. 1981. Health and Ecological Assessment of Polynuclear Aromatic Hydrocarbons. Pathotox Publishers, Inc., Park Forest South, IL.

Schwarz, F.P. and S.P. Wasik. 1976. Fluorescence measurements of benzene, naphthalene, anthracene, pyrene, fluoranthene and benzo(e)pyrene in water. Anal. Chem. 48: 524-528.

Scribner, J.D. 1973. Brief communication. Tumor initiation by apparently noncarcinogenic polycyclic aromatic hydrocarbons. J. Natl. Cancer Inst. 50: 1717-1719.

Sellakumar, A. and P. Shubik. 1974. Carcinogenicity of different polycyclic hydrocarbons in the respiratory tract of hamsters. J. Natl. Cancer Inst. 53: 1713-1719.

Shear, M.J. and J. Leiter. 1941. Studies in carcinogenesis. XVI. Production of subcutaneous tumors on mice by miscellaneous polycyclic compounds. J. Natl. Cancer Inst. 11: 241-258.

Shimada, T., K. Furukawa, D.M. Kreiser, A. Cawein and G.M. Williams. 1983. Induction of transformation by six classes of chemical carcinogens in adult rat liver epithelial cells. Cancer Res. 43(11): 5087-5092.

Sims, P. 1970. Qualitative and quantitative studies on the metabolism of a series of aromatic hydrocarbons by rat-liver preparations. Biochem. Pharmacol. 19: 795-818.

Sims, R.C. and M.R. Overcash. 1983. Fate of polynuclear aromatic compounds (PNAS) in soil-plant systems. Residue Reviews. 88: 1-68.

Sina, J.F., C.L. Bean, G.R. Dysart, V.I. Taylor, M.O. Bradley. 1983. Evaluation of the alkaline elution/rat hepatocyte assay as a predictor of carcinogenic/mutagenic potential. Mutat. Res. 113(5): 357-391.

Sorrell, R.K., H.J. Brass and R. Reding. 1980. A review of the occurrences and treatment of polynuclear aromatic hydrocarbons in water. Environ. Inter. 4: 245-254.

Southworth, G.R., J.J. Beauchamp and P.K. Schmieder. 1978. Bioaccumulation potential of polycyclic aromatic hydrocarbons in Daphnia pulex. Water Res. 12: 973-977.

Spehar, R.L., R.W. Carlson, A.E. Lemke, D.I. Mount, Q.H. Pickering and V.M. Snarski. 1980. Effects of pollution on freshwater fish. J. Water Pollut. Control Fed. 52: 1703-1774.

SRI (Stanford Research Institute). 1986. 1986 Directory of Chemical Producers, United States of America. SRI International, Menlo Park, CA.

Staples, C.A., A. Werner and T. Hoogheem. 1985. Assessment of priority pollutant concentrations in the United States using STORET database. Environ. Toxicol. Chem. 4: 131-142.

Swann, R.L., D.A. Laskowski, P.J. McCall, K.VanderKuy and H.J. Dishburger. 1983. A rapid method for the estimation of the environmental parameters octanol/water partition coefficient, soil sorption constant, water to air ratio, and water solubility. Res. Rev. 85: 17-28.

Tabak, H.H., S.A. Quave, C.I. Mashni and E.F. Barth. 1981. Biodegradability studies with organic priority pollutant compounds. J. Water Pollut. Control Fed. 53: 1503-1518.

Tan, Y.L. and M. Heit. 1981. Biogenic and abiogenic polynuclear aromatic hydrocarbons in sediments from two remote Adirondack lakes. Geochim. Cosmochim. Acta. 45: 2267-2279.

Topham, J.C. 1980. The detection of carcinogen-induced sperm head abnormalities in mice. Mutat. Res. 69(1): 149-155.

Topping, D.C., B.C. Pal, D.H. Martin, F.R. Nelson and P. Nettesheim. 1978. Pathologic changes induced in respiratory tract mucosa by polycyclic hydrocarbons of differing carcinogenic activity. Am. J. Pathol. 93: 311-324.

U.S. EPA. 1977. Computer print-out of non-confidential production data from TSCA inventory. OPTS, CID, U.S. EPA, Washington, DC.

U.S. EPA. 1980. Ambient Water Quality Criteria for Polynuclear Aromatic Hydrocarbons. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Water Regulations and Standards, Washington, DC. EPA 440/5-80-069. NTIS PB 81-117806.

U.S. EPA. 1983. Reportable Quantity for Pyrene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, OHEA, for the Office of Emergency and Remedial Response, Washington, DC.

U.S. EPA. 1984. Health Effects Assessment for Pyrene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Emergency and Remedial Response, Washington, DC. EPA 540/1-86-030.

U.S. EPA. 1986a. Computer printout: Graphic Exposure Modeling System (GEMS) EXAMS model. U.S. EPA, Office of Toxic Substances, Washington, DC.

U.S. EPA. 1986b. Guidelines for carcinogenic risk assessment. Federal Register. 51(185): 3392-34003.

U.S. EPA/NIH. 1986. OHM-TADS (Oil and Hazardous Materials Technical Assistance Data System). April 16, 1986: On-line.

Vaessen, H.A.M.G., P.L. Schuller, A.A. Jekel and A.A.M.M. Wilbers. 1984. Polycyclic aromatic hydrocarbons in selected foods. Analysis and occurrence. Toxicol. Environ. Chem. 7: 297-324.

Valencia, R. and K. Houtchens. 1981. Mutagenic activity of 10 coded compounds in the Drosophila sex-linked recessive lethal test. In: Evaluation of Short-Term Tests for Carcinogens. Report of the International Collaborative Program. Progress in Mutation Research, F.J. deSerres and J. Ashby, Ed. Elsevier/North Holland, NY. Vol. 1, p. 651-659.

Valerio, F. and A. Lazzarotto. 1984. Role of airborne particulate matter components on photochemical degradation of pyrene, benzoanthracene and benzo(a)pyrene. A preliminary report. Spectrosc. Int. J. 3: 412-415.

Van Duuren, B.L. and B.M. Goldschmidt. 1976. Cocarcinogenic and tumor-promoting agents in tobacco carcinogenesis. J. Natl. Cancer Inst. 56: 1237-1242.

Van Noort, P.C.M. and E. Wondergem. 1985. Scavenging of airborne polycyclic aromatic hydrocarbons by rain. Environ. Sci. Technol. 19: 1044-1048.

Varanasi, U., W.L. Reichert, J.E. Stein, D.W. Brown and H.R. Sanborn. 1985. Bioavailability and biotransformation of aromatic hydrocarbons in benthic organisms exposed to sediment from an urban estuary. Environ. Sci. Technol. 19: 836-841.

Ved Brat, S., C. Tong, S. Teland and G.M. Williams. 1983. Comparison of sister chromatid exchange and mammalian cell mutagenesis at the hypoxanthine guanine phosphoribosyl transferase locus in adult rat liver epithelial cells. Ann. N.Y. Acad. Sci. 407: 474-475.

Walker, J.D. and R.R. Colwell. 1975. Degradation of hydrocarbons and mixed hydrocarbon substrate by microorganisms from Chesapeake Bay. Prog. Water Technol. 7: 783-791.

Walters, R.W. and R.G. Luthy. 1984. Liquid/suspended solid phase partitioning of polycyclic aromatic hydrocarbon in coal coking waste waters. Water Res. 18: 795-809.

Weast, R.C., Ed. 1980. CRC Handbook of Chemistry and Physics, 61st ed. CRC Press, Inc., Boca Raton, FL. p. C-527.

Weaver, N.K. and R.L. Gibson. 1979. The U.S. oil shale industry: A health perspective. Am. Ind. Hyg. Assoc. J. 40(6): 460-467.

White, J. and A. White. 1939. Inhibition of growth of the rat by oral administration of methylcholanthrene, benzpyrene, or pyrene and the effects of various dietary supplements. J. Biol. Chem. 131: 149-161.

Williams, D.T., E.R. Nestmann, G.L. Lebel, F.M. Benoit, R. Otson and E.G.H. Lee. 1982. Determination of mutagenic potential and organic contaminants of Great Lakes drinking water. Chemosphere. 11: 263-276.

Windholz, M., Ed. 1983. The Merck Index, 10th ed. Merck and Co., Inc., Rahway, NJ. p. 1148-1149.

Wood, A.W., W. Levin, R.L. Chang, et al. 1980. Mutagenicity and tumor-initiating activity of cyclopenta(3,d)pyrene and structurally related compounds. Cancer Res. 40(3): 642-649.

Wynder, E.L. and D. Hoffman. 1959. A study of tobacco carcinogens. VII. The role of higher polycyclic hydrocarbons. Cancer. 12: 1079-1086.

Yalkowsky, S.H. and S.C. Valvani. 1979. Solubilities and partioning. 2. Relationships between aqueous solubilities, partition coefficients, and molecular surface areas of rigid aromatic hydrocarbons. J. Chem. Eng. Data. 24: 127-129.

Yamasaki, H., K. Kuwata and H. Miyamoto. 1982. Effects of ambient temperature on aspects of airborne polycyclic aromatic hydrocarbons. Environ. Sci. Technol. 16: 189-194.

Yokley, R.A., A.A. Garrison, E.L. Wehry and G. Mamantor. 1986. Photochemical transformation of pyrene vapor-deposited on eight coal stack ashes. Environ. Sci. Technol. 20: 86-90.

Yoshikawa, T., L.P. Ruhr, W. Flory, D. Giamalva, D.F. Church and W.A. Pryor. 1985. Toxicity of polycyclic aromatic hydrocarbons. I. Effect of phenanthrene, pyrene and their ozonized products on blood chemistry in rats. Toxicol. Appl. Pharmacol. 79(2): 218-226.

Zepp, R.G. and P.F. Schlotzhauer. 1983. Influence of algae on photolysis rates of chemicals in water. Environ. Sci. Technol. 17: 462-468.

Zepp, R.G., P.F. Schlotzhauer, M.S. Simmons, G.C. Miller, G.L. Baughman and N.L. Wolfe. 1984. Dynamics of pollutant photoreactions in the hydrosphere. Fresenius Z. Anal. Chem. 319: 119-125.

APPENDIX

LITERATURE SEARCHED

This profile is based on data identified by computerized literature searches of the following:

GLOBAL
TSCATS
CASR online (U.S. EPA Chemical Activities Status Report)
CAS online STN International
TOXLINE
TOXBACK 76
TOXBACK 65
RTECS
OHM TADS
STORET
SRC Environmental Fate Data Bases
SANSS
AQUIRE
TSCAPP
NTIS
Federal Register

These searches were conducted in April, 1986. In addition, hand searches were made of Chemical Abstracts (Collective Indices 6 and 7), and the following secondary sources were reviewed:

ACGIH (American Conference of Governmental Industrial Hygienists). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices, 5th ed. Cincinnati, OH.

ACGIH (American Conference of Governmental Industrial Hygienists). 1985-1986. TLVs: Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environment with Intended Changes for 1985-1986. Cincinnati, OH. 114 p.

Clayton, G.D. and F.E. Clayton, Ed. 1981. Patty's Industrial Hygiene and Toxicology, 3rd rev. ed., Vol. 2A. John Wiley and Sons, NY. 2878 p.

Clayton, G.D. and F.E. Clayton, Ed. 1981. Patty's Industrial Hygiene and Toxicology, 3rd rev. ed., Vol. 2B. John Wiley and Sons, NY. p. 2879-3816.

Clayton, G.D. and F.E. Clayton, Ed. 1982. Patty's Industrial Hygiene and Toxicology, 3rd rev. ed., Vol. 2C. John Wiley and Sons, NY. p. 3817-5112.

Grayson, M. and D. Eckroth, Ed. 1978-1983. Kirk-Othmer Encyclopedia of Chemical Technology, 3rd ed. John Wiley and Sons, NY. 23 Volumes.

Hamilton, A. and H.L. Hardy. 1974. Industrial Toxicology, 3rd ed. Publishing Sciences Group, Inc., Littleton, MA. 575 p.

IARC (International Agency for Research on Cancer). IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans. WHO, IARC, Lyons, France.

ITII (International Technical Information Institute). 1982. Toxic and Hazardous Industrial Chemicals Safety Manual for Handling and Disposal with Toxicity and Hazard Data. ITII, Tokyo, Japan. 700 p.

Jaber, H.M., W.R. Mabey, S.T. Liu, T.W. Chow and H.L. Johnson. 1984. Data aquisition for environmental transport and fate screening for compounds of interest in the Office of Solid Waste. EPA 600/6-84-010. NTIS PB84-243906. SRI International, Menlo Park, CA.

NTP (National Toxicology Program). 1986. Toxicology Research and Testing Program. Chemicals on Standard Protocol. Management Status.

Ouellette, R.P. and J.A. King. 1977. Chemical Week Pesticide Register. McGraw-Hill Book Co., NY.

Sax, N.I. 1979. Dangerous Properties of Industrial Materials, 5th ed. Van Nostrand Reinhold Co., NY.

SRI (Stanford Research Institute). 1984. Directory of Chemical Producers. Menlo Park, CA.

U.S. EPA. 1985. Status Report on Rebuttable Presumption Against Registration (RPAR) or Special Review Process. Registration Standards and the Data Call in Programs. Office of Pesticide Programs, Washington, DC.

U.S. EPA. 1985. CSB Existing Chemical Assessment Tracking System. Name and CAS Number Ordered Indexes. Office of Toxic Substances, Washington, DC.

USITC (U.S. International Trade Commission). 1983. Synthetic Organic Chemicals. U.S. Production and Sales, 1982, USITC Publ. 1422, Washington, DC.

Verschueren, K. 1983. Handbook of Environmental Data on Organic Chemicals, 2nd ed. Van Nostrand Reinhold Co., NY.

Windholz, M., Ed. 1983. The Merck Index, 10th ed. Merck and Co., Inc., Rahway, NJ.

Worthing, C.R. and S.B. Walker, Ed. 1983. The Pesticide Manual. British Crop Protection Council. 695 p.

In addition, approximately 30 compendia of aquatic toxicity data were reviewed, including the following:

Battelle's Columbus Laboratories. 1971. Water Quality Criteria Data Book. Volume 3. Effects of Chemicals on Aquatic Life. Selected Data from the Literature through 1968. Prepared for the U.S. EPA under Contract No. 68-01-0007. Washington, DC.

Johnson, W.W. and M.T. Finley. 1980. Handbook of Acute Toxicity of Chemicals to Fish and Aquatic Invertebrates. Summaries of Toxicity Tests Conducted at Columbia National Fisheries Research Laboratory. 1965-1978. U.S. Dept. Interior, Fish and Wildlife Serv. Res. Publ. 137, Washington, DC.

McKee, J.E. and H.W. Wolf. 1963. Water Quality Criteria, 2nd ed. Prepared for the Resources Agency of California, State Water Quality Control Board. Publ. No. 3-A.

Pimental, D. 1971. Ecological Effects of Pesticides on Non-Target Species. Prepared for the U.S. EPA, Washington, DC. PB-269605.

Schneider, B.A. 1979. Toxicology Handbook. Mammalian and Aquatic Data. Book 1: Toxicology Data. Office of Pesticide Programs, U.S. EPA, Washington, DC. EPA 540/9-79-003. NTIS PB 80-196876.